

THE EPIDEMIOLOGY OF BLUE MOLD OF TOBACCO AND ITS RELATION TO THE INCIDENCE OF THE DISEASE IN ONTARIO<sup>1</sup>R. H. STOVER AND L. W. KOCH<sup>2</sup>*Laboratory of Plant Pathology, Harrow, Ont.*

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## INTRODUCTION

From 1938, the year of the initial occurrence of blue mold (*Peronospora tabacina* Adam) of tobacco in Canada, until 1945, only sporadic occurrences of this disease were reported in the tobacco-growing districts of Ontario. Then suddenly in 1945, an epidemic of blue mold occurred in southern Ontario. Scarcely a seedbed escaped infection and many were destroyed. Again, in 1946, the disease reached epidemic proportions, causing widespread damage to seedlings and, in some localities, to plants in the field. Since 1946, the disease has been less prevalent and, with each succeeding year, there has been a tendency for growers to take fewer precautions against possible epidemics. Thus, the question arises as to whether or not precautions are necessary, and, if so, when they should be implemented. To attempt to answer these questions, it is obviously necessary to make a comprehensive study of conditions that contribute towards, or are the cause of blue mold epidemics in Ontario. Since climatic conditions and the source of inoculum are probably the most important factors involved, the present study was undertaken to determine their role in the epidemiology of this disease in Ontario.

## REVIEW OF LITERATURE

*Climate in Relation to Development of the Disease*

Dixon *et al.* (9), in 1936, concluded that primary outbreaks resulting from oospore germination occur during or immediately after periods of warm weather, with the minimum temperature of the surface layer of soil being maintained at or above 50° F. for several days to allow germination of oospores. These investigators also found sporulation to be favoured by long periods of atmospheric saturation when the sky is overcast. Favourable temperatures for sporulation ranged from 42° to 63° F., with the optimum at 56° F. At temperatures above 63° F. or below 42° F., sporulation was observed to be sparse. Few sporangia were formed above 68° F. or below 36° F.

Clayton and Gaines (5) found that sporulation did not occur unless the temperature dropped below 70° F. Their experiments also showed that night temperatures of from 55° to 60° F. favour sporulation, but that

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alternating higher and lower temperatures were much more favourable for sporulation than a uniform lower temperature. Spore germination occurred over a wide range of temperatures, varying with different spore collections. No germination of spores was obtained at 85° or above, or at 32° F. or below. Wolf (39) states that temperatures between 45° and 65° F. are most favourable for spore germination. Armstrong and Sumner (3), in 1935, reported some sporulation at temperatures above 80° F.; Clayton and Gaines (5), in 1945, found blue mold developing freely, and producing spores as well, at temperatures from 70° to 80° F., and they reported that optimum temperatures were higher for infection than for sporulation. Free moisture is apparently essential for both germination and infection (4, 23).

Clayton (4), in 1939, stated that in early spring, with minimum night temperatures ranging from 40° to 50° F., the disease does not develop rapidly. Outbreaks assume epidemic proportions with the advent of warmer nights, usually with a minimum of 50° F. or higher, accompanied by fogs or light rains. Heavy rains tend to check development of the causal organism, and, as the minimum night temperature rises to 65° or 70° F., the disease disappears. The mycelium is very active at 75° F., but the leaf tissue dies rapidly and few, if any, spores are produced. Night temperatures of approximately 56° F., with dew or rain for from two to three hours after sunrise, provide ideal conditions for germination and infection (23). Few sporangia are developed at a night temperature above 68° F.

Viability of sporangia is conditioned by temperature, moisture, and sunshine (5, 25). Sporulation occurs most abundantly shortly after dawn, and, by 11.00 a.m., most of the spores are dead (5). Wolf (25) reported finding approximately 1 per cent of the sporangia viable after from four hours to seven days, and concluded that longevity is correlated with low temperatures. Exposure to temperatures of approximately 85° F. for an hour is lethal, as is exposure to direct sunlight for a like period (23, 25). Newhall (14), in 1938, concluded that sunshine in itself is not lethal to conidia of *Peronospora* if they do not become dry or too warm. Clayton (4), in 1939, stated that most of the spores are dead by 9.00 a.m., but a few may remain alive under favourable conditions for as long as two weeks. Certain instances have been reported (5, 25) in which spores have remained viable for very long periods. Wolf (25) reported that in Australia sporangia remained viable under moist conditions at 18° C. for two months. Temperatures as low as 16° C. have been reported by Clayton and Gaines (5) as being favourable for the storage of sporangia.

Recovery from blue mold is assisted by warm, bright days, with maximum daytime temperatures of approximately 90° F. and night temperatures which do not fall below 68° F. (25). Dixon *et al.* (9) stated that although factors within the plant are of primary importance in recovery yet recovery is more rapid during warm clear days and warm nights. Clayton and Gaines (5) reported that high temperature infections are possible under conditions of low light intensity, coupled with tender, succulent growth.

### *Source of Inoculum and Means of Dissemination*

In certain areas, oospores are formed in the dead leaf tissue within a few days after the leaves are killed. Little is known, however, about the factors determining their formation and germination. According to Wolf (24), oospores have been collected in Connecticut, Maryland, North Carolina, South Carolina, and Virginia, and they may be presumed to occur wherever the pathogen exists.

Dixon *et al.* (10) regard primary infections as being those which occur in seedbeds sown on the sites of old diseased seedbeds if (i) sporulation occurs in such beds from 9 to 17 days prior to sporulation in any new seed bed in the locality, if (ii) when sporangia are produced, sporulation occurs in a small area in the seedbed on the leaves of a few seedlings, and if (iii) oospores constitute the inoculum for these infections. Furthermore, these investigators state that, if oospores constitute the source of inoculum for primary infections, they must be in the surface layer of soil, and the maintenance of satisfactory temperature levels (50° F.) in the surface soil for a sufficient length of time is necessary for germination.

As the sporangial cycle is repeated every four to seven days, a seedbed infected originally from oospores of the causal organism can, within three weeks, provide sufficient inoculum to initiate an outbreak involving all seedbeds in an area of several hundred square miles (24). Dixon *et al.* (10) noted that secondary infections are predominant in the locality surrounding a primary-infected seedbed within two weeks after primary infection appears. Sporulation continues in all seedbeds during cloudy, wet weather for a period of from ten to fifteen days. During or after that period, the disease becomes general over the whole district (10). Weather factors affect these conditions to the extent of lengthening the period of the secondary outbreak, or shortening the period between the beginning of the secondary outbreak and a general infection (10, 24).

Overwintering of the fungus on volunteer tobacco plants and suckers in Georgia and Florida is considered common (18, 20, 25). There is also a possibility, according to Dixon *et al.* (10), of this type of overwintering in North Carolina. Valleau (20, 22) states that the fungus can maintain itself in Kentucky or Tennessee only if spores are blown in from the south, and that the same would probably hold true in the Carolinas and Virginia only if spores are blown in from the Georgia-Florida area. From his study of the incidence of primary infections in seedbeds located on the site of old seedbeds previously infected, Wolf (27) concludes that the fungus overwinters in the oospore stage, and that this stage serves as the inoculum for primary infections in old plant beds in North Carolina.

Kighlinger (13) states that the disease was probably introduced into the Connecticut Valley by means of windblown spores, but he believes that subsequently infections have originated from overwintering oospores. Valleau (21) indicates that the 1946 outbreak in Kentucky originated from oospores in second-year seedbeds, whereas the 1945 outbreak originated from spores blown in from the southeast. Clayton (7) observed that overwintering of oospores in the north is not so common as in the south, and that primary infections in Maryland are usually due to a spore shower. Wolf (28) claims that oospores are the primary source of inoculum through-

out the entire region of the Carolinas and indicates that Anderson, in Connecticut, reached the same conclusion about oospores for that area. Wolf (24) noted that an outbreak originating from oospores usually occurs by the time the seedlings have reached the four-leaf stage, and, after the first spell of warm weather, a general outbreak follows in from three to four weeks.

The most important agency for the dissemination of blue-mold spores is indicated to be air currents (9, 10, 17). Wolf *et al.* (26) trapped air-borne spores on glycerine-coated glass plates in the vicinity of diseased seedbeds. They report the dissemination period between primary and secondary outbreaks to be from 12 to 17 days, and between the secondary and general outbreak, 6 to 12 days. The amount of inoculum trapped depended upon the amount of sporulation which, in turn, was dependent upon weather conditions and wind.

The rapid spread of the disease, as related by Stevens and Wood (18), can only be explained by dissemination occurring through air-borne sporangia. Anderson (1) explains the sudden and widespread appearance of the disease in Connecticut within less than a week after the first infection was seen in 1937, as primary infections due to wind-blown spores from Pennsylvania or the states to the south. According to Clayton (4), blue mold spores are readily carried 50 to 75 miles or more by air currents, and unprotected seedbeds in an infected area are unlikely to escape the disease.

Dixon *et al.* (10) observed that, although continued rainy weather, combined with long periods of atmospheric saturation, was conducive to sporulation, such conditions inhibited dissemination. They account for the wave-like northward advance of the disease in the southern states as being associated with difference in time of occurrence of primary infections, in the different localities, rather than to sporangia being transported to these localities.

From the foregoing review of literature, it is apparent that conflicting views exist as to the relative importance of oospores and/or air-borne conidia as sources of inoculum which give rise to epidemics in Kentucky, Connecticut, and other northern states.

#### *History and Prevalence of the Blue Mold Disease of Tobacco in the United States*

The blue mold disease of tobacco was first recorded in the United States in 1921 in Gadsden County, Florida, and Decatur County, Georgia (25). At the time, two theories were suggested as to the origin of the disease (16), namely, that it had either recently been introduced, probably from Australia, or that it had become abundant in the southeastern United States after migrating from the western part of the country. With regard to the latter theory it may be mentioned that, according to Stevens and Ayres (16), a downy mildew, probably *P. tabacina*, was collected by Wilson on *Nicotiana bigelovii*, in Nevada in 1914. The disease did little damage in the first year of its appearance, and disappeared for ten consecutive years after the 1921 outbreak.

The 1931 outbreak of blue mold was more serious. Not only did the disease again appear in Georgia and Florida in the same areas as in 1921, but it later spread to the flue-cured tobacco areas of Georgia, North

Carolina, and Virginia, throughout the entire coastal plains and Piedmont sections of the Atlantic seaboard, and into the air-cured tobacco region of southern Maryland (9, 18). The occurrence of blue mold on this occasion was considerably earlier in the season than in the case of the 1921 outbreak.

Reasons for the disappearance of the disease from 1921 to 1931 were not correlated with weather conditions, but rather with the fact that oospores were not present in any of the material examined. Therefore, little or no carry-over of the fungus was possible in 1921 (6). Stevens and Ayres (16) stated that the most reasonable conclusion was that the outbreak of 1931 represented a new introduction, separate and distinct from the 1921 outbreak. Evidence presented by Valleau (22) and Wolf (29) indicates this introduction probably originated from wild *Nicotiana* species in Texas.

In 1932, the disease extended its range in Florida, Georgia, South Carolina, North Carolina, and Virginia. It also spread to the burley area of central Virginia, and recurred in southern Maryland (16, 25, 27). Stevens and Wood (18) state that it was found in Lancaster County, Pennsylvania in 1932. Losses in the Carolinas from blue mold were very heavy in 1932 (16, 18).

Stevens and Wood (18) consider 1933 as the "climax year" for the spread of blue mold. That year it spread farther into the burley areas of southwestern Virginia (1, 18), western North Carolina, and eastern and central Tennessee, and became more widespread in the states in which it had previously occurred.

During 1934 and 1935 the disease was less severe, and Wolf (27) records less damage than in any other year during the planting season of 1934 to North Carolina. Stevens and Wood (18) report blue mold for 1934 and again occurring in Lancaster County, Pennsylvania. It was found in two new counties in 1935, in Tennessee. The damage and severity increased slightly in 1935, but was not as severe as in 1933 (16, 18, 27).

The disease was reported for the first time in Kentucky in 1936, but was not prevalent (17). Little damage was experienced during the 1936 planting season in any of the United States tobacco-growing areas (16).

In 1937 blue mold appeared again in Kentucky, and no seedbeds escaped infection, except those in the extreme western part of the state (20). The disease was severe in the more southerly states (16, 17). Serious damage in the field occurred for the first time, with losses in Georgia up to 50 per cent, and in Florida from 10 to 35 per cent (16, 17). The disease appeared for the first time in Indiana, the Connecticut Valley, and southern Massachusetts in 1937 (1, 17, 24).

In 1938 blue mold appeared in Ohio for the first time, and was widespread (11). Only moderate damage was reported from Pennsylvania in 1938. In the same year the disease was reported for the first time in Essex County, Canada (8). Stevens (18) states that blue mold reached Franklin, Massachusetts, the northernmost section of the tobacco-growing area in 1938. Since 1938, the distribution of the disease on tobacco has remained constant, with the exception of its appearance in Wisconsin in 1947 (12). After the 1937 and 1938 outbreaks, blue mold seemed to decline in severity in most of the tobacco-growing states or, at least, it became no

worse than the 1937 and 1938 epidemics. During the period 1939 to 1944, the incidence of the disease decreased greatly in both Kentucky and Tennessee.

According to Janson (11), Ohio was virtually free from blue mold from 1940 until 1944.

In Pennsylvania, Street (19) states that the disease was of minor importance for a considerable number of years after its initial occurrence in 1932 or 1933. However, by 1943, blue mold was firmly established in Pennsylvania and in the following year the disease was prevalent throughout the state. In 1940, approximately one-quarter of the seedbeds were destroyed by blue mold in Pennsylvania (Table 1).

In Massachusetts and in Connecticut, Kighlinger (13) recorded blue mold as occurring rather commonly in seedbeds in June, 1943, but it was not so prevalent as usual and did little damage. The lightest attack of blue mold in these states occurred in 1944.

In 1945\*, in the more southerly states, blue mold was no more serious than in previous years. The outbreak was considered a little more severe in South Carolina and Tennessee. In the Connecticut Valley, including Massachusetts, the infection as a whole was extremely light. In the area including western North Carolina, Virginia, Maryland, Pennsylvania, and Ohio, the outbreak was severe, the most severe ever experienced in the latter two states. In Kentucky the disease was widespread and caused severe injury.

In 1946, reports from Kentucky indicated the most severe outbreak experienced to date in that state, with Ohio and Pennsylvania also recording a widespread and severe attack.

In 1947, Georgia and eastern North Carolina experienced a severe epidemic, but only mild damage occurred in the other tobacco-growing states. In the following year, most areas experienced light to moderate attacks of the disease. This was particularly true for Kentucky, Connecticut, North Carolina, and Pennsylvania.

In 1949, damage was slight, except in the Carolinas and Virginia which experienced severe injury. Reports for this year indicated only scattered occurrences of the disease in Kentucky and other areas, with none from Ohio and very few from Pennsylvania and Massachusetts.

A summary of the data relating to the incidence of the disease in Ohio, Kentucky and Pennsylvania is given in Table 1. The importance of these areas as a source of inoculum for Ontario outbreaks will be discussed in another section.

#### **OCCURRENCE OF THE BLUE MOLD DISEASE OF TOBACCO IN ONTARIO**

Before the occurrence of blue mold in Ontario is discussed in detail, it should be mentioned that there are two distinct tobacco-growing districts in the province. The part usually referred to as the Old Tobacco Belt consists of Essex and Kent counties, at the extreme southwestern end of the province, whereas the New Tobacco Belt consists of Norfolk and surrounding counties 100 miles or more east of the Old Belt (Figure 1).

\* Data based on information in The U.S.D.A. Plant Disease Reporter and Warning Service circulars.

TABLE 1.—DATA RELATING TO THE INCIDENCE OF BLUE MOLD OF TOBACCO IN PENNSYLVANIA, OHIO AND KENTUCKY FROM 1938 TO 1950

| Year | Pennsylvania       |                  |                  | Ohio               |                  |                  | Kentucky             |                  |                  |
|------|--------------------|------------------|------------------|--------------------|------------------|------------------|----------------------|------------------|------------------|
|      | Date of appearance | Extent of spread | Degree of injury | Date of appearance | Extent of spread | Degree of injury | Date of appearance   | Extent of spread | Degree of injury |
| 1936 | —                  | —                | —                | —                  | —                | —                | June 8*              | Limited          | Mild             |
| 1937 | —                  | —                | —                | —                  | —                | —                | May 13               | Widespread       | Severe           |
| 1938 | ?                  | —                | Mild             | June 13*           | Sporadic         | Mild             | April 29             | General          | Moderate         |
| 1939 | ?                  | Limited          | Mild             | May 23             | Limited          | Mild             | May 7                | Limited          | Mild             |
| 1940 | May 12             | Widespread       | Severe           | —                  | —                | —                | May 15               | Limited          | Mild             |
| 1941 | —                  | Limited          | Mild             | June 13            | Limited          | Mild             | —                    | Limited          | Mild             |
| 1942 | —                  | Limited          | Mild             | —                  | —                | —                | —                    | Limited          | Mild             |
| 1943 | —                  | Limited          | Mild             | —                  | —                | —                | —                    | Limited          | Mild             |
| 1944 | —                  | Widespread       | Moderate         | —                  | —                | —                | —                    | —                | —                |
| 1945 | May 22             | Widespread       | Severe           | June 2             | Widespread       | Severe           | April 28** to May 11 | Widespread       | Severe           |
| 1946 | May 10             | Widespread       | Severe           | May 1              | Widespread       | Severe           | April 17** to May 2  | Widespread       | Severe           |
| 1947 | May 20             | Limited          | Mild             | May 17             | Widespread       | Moderate         | May 8                | Widespread       | Moderate         |
| 1948 | May 17             | Limited          | Mild             | —                  | —                | —                | April 29             | Widespread       | Moderate         |
| 1949 | ?                  | Limited          | Mild             | —                  | Limited          | Mild             | May 2                | Limited          | Mild             |
| 1950 | ?                  | Limited          | Mild             | —                  | —                | —                | May 5                | Limited          | Mild             |

\* Initial occurrence in state.

\*\* The two dates represent the difference in the date of disease occurrence at the south and north State boundaries, respectively.

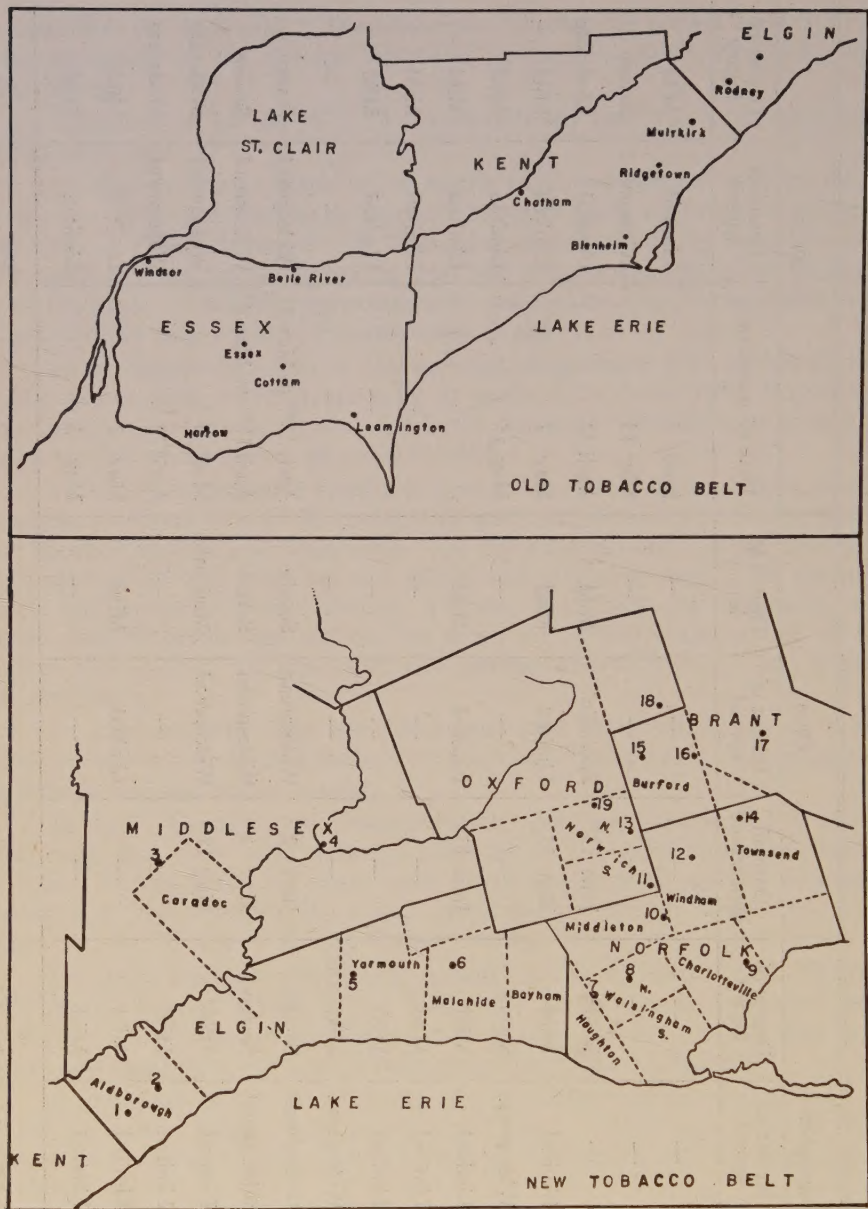


FIGURE 1. Map of the Old (*upper*) and the New (*lower*) Tobacco Belts of Ontario. Burley tobacco is grown in the Old Belt, whereas flue-cured tobacco is grown almost exclusively in the much larger New Belt. The greatest concentration of flue-cured tobacco in Canada is in Norfolk County. All locations mentioned in the text are marked on these maps. Numbers in the New Belt designate the following locations: 1, Rodney; 2, West Lorne; 3, Strathroy; 4, London; 5, St. Thomas; 6, Aylmer; 7, Glen Meyer; 8, Langton; 9, Vittoria; 10, Delhi; 11, LaSalette; 12, Teeterville; 13, Norwich; 14, Wilsonville; 15, Cathcart; 16, Burford; 17, Brantford; 18, Prince-ton; 19, Burgessville.

TABLE 2.—DATA CONCERNING THE OCCURRENCE AND PREVALENCE OF BLUE MOLD OF TOBACCO IN ONTARIO FROM 1938 TO 1950

| Year | Old Tobacco Belt                   |                                               | New Tobacco Belt        |                                                                                          |
|------|------------------------------------|-----------------------------------------------|-------------------------|------------------------------------------------------------------------------------------|
|      | First appearance                   | Incidence and severity                        | First appearance        | Incidence and severity                                                                   |
| 1938 | June 25, Essex*                    | Widespread but moderate<br>Harrow to Blenheim | Nil                     | —                                                                                        |
| 1939 | June 20, N.W. of Essex             | Eight seedbeds, 2 fields,<br>moderate         | Nil                     | —                                                                                        |
| 1940 | July 5, Blenheim                   | Limited spread, mild                          | Nil                     | —                                                                                        |
| 1941 | Nil                                | —                                             | Nil                     | —                                                                                        |
| 1942 | Nil                                | —                                             | Nil                     | —                                                                                        |
| 1943 | June 18, Belle River,<br>Harrow    | Limited spread, Essex<br>Co., mild            | Nil                     | —                                                                                        |
| 1944 | June 9, Harrow                     | Limited spread, Essex<br>Co., mild            | Nil                     | —                                                                                        |
| 1945 | June 8, Essex and Kent<br>Counties | Epidemic                                      | June 10, Norfolk County | Epidemic                                                                                 |
| 1946 | May 20, Chatham,<br>Rodney         | Epidemic                                      | May 18, Wilsonville     | Epidemic                                                                                 |
| 1947 | May 11, Leamington                 | Widespread and severe                         | May 26, Cathcart        | Widespread and severe                                                                    |
| 1948 | May 25, Cottam                     | Sporadic, mild                                | May 17, Norwich         | Sporadic but severe in<br>some greenhouses.<br>Considerable field in-<br>fection in July |
| 1949 | June 3                             | Sporadic, mild                                | May 15, Princeton       | Sporadic in greenhouses.<br>Considerable field in-<br>fection                            |
| 1950 | June 1                             | Sporadic, mild                                | May 11, Langton         | Sporadic in greenhouses.<br>Widespread in fields                                         |

\* For locations, see Figure 1.

As indicated earlier, blue mold first appeared on tobacco in Ontario in 1938. It was observed in 8 seedbeds and 4 fields (8). The first infections appeared near the town of Essex; later infections were reported from Harrow and Blenheim (Figure 1, Table 2).

In 1939, the disease made its appearance on two farms near Essex, in the same district as in the previous year. Eight seedbeds and 2 fields were involved, and it appeared first in seedbeds in which it had been present in the previous year. Koch (8) suggested that the fungus probably overwintered.

In 1940, blue mold occurred in a single seedbed at Blenheim, Kent County, on a farm where the disease had destroyed all the seedbeds in 1938, but did not appear in 1939. Quite possibly either the pathogen may have survived on hosts other than tobacco or the disease may have been present but unnoticed in 1939, or oospores, if present, may have remained dormant throughout one plantbed season.

In 1941 and 1942, blue mold was not found in Ontario (8). In 1943, it was reported near Harrow and Belle River, two areas 35 miles apart in Essex County (Table 2). During this year, the disease was present in one field where transplanting has been completed before the disease was diagnosed and high temperatures had inhibited further development. In 1944, no infection was found, except in one seedbed near Harrow, Ontario. Then, in 1945 and again in 1946, a destructive epidemic occurred throughout the entire tobacco-growing areas of southern Ontario bordering on Lake Erie (Figure 1).

The initial outbreaks in 1945 were reported on June 8 from widely-separated localities in both the Old and the New Tobacco Belt. On June 11 and 12, the disease was present in Malahide and Charlotteville Townships (see Figure 1); on June 16, it was general in Caradoc, Walsingham, Middleton, and Windham Townships in the New Belt, and in most of the Old Belt districts. Eventually the epidemic affected approximately 95 per cent of the seedbeds in the Old Belt. A field outbreak occurred during the period July 13 to 16. Field infections were widespread as far north as Burgessville, Norwich Township. Severe seedling injury was caused throughout the New Belt and also in some fields in the Duart, Ridgetown and Harrow districts. The fungus was found fruiting on field plants near Burford as late as August 14.

In 1946, the first reports of blue mold were received from Wilsonville on May 18, several weeks earlier than those for any previous year. This outbreak occurred in a greenhouse in an area where presence of the disease was not recorded in the previous year. No other diseased seedbeds were found in the area at that time. On May 20, the disease was present in the Chatham and Rodney districts, on May 27 in Burford Township, and on May 31 in Caradoc Township. On June 6, several reports of blue mold were received from Bayham Township and the LaSalette district. By June 8, the disease was present in Charlotteville, Walsingham, Houghton, and Middleton Townships of the New Belt, and it was widespread in the Old Belt. Because of the early transplanting season in the New Belt, and the extensive spraying and dusting program, damage was not so extensive as it might have been had the seedbeds not been so far advanced and protected. By June 1, almost 50 per cent of the crop was planted, and, by June 8, more than 90 per cent was in the fields. In some districts, many planters experienced an acute shortage of plants because of the destruction of numerous seedbeds. By the end of the season, nearly all seedbeds were infected.

In 1947, blue mold made its appearance in the Leamington district, in the Old Belt, on May 11 and in Oxford and Norfolk counties in the New Belt, on May 26. During June, the disease reached epidemic proportions in Essex and Norfolk counties. It caused considerable damage in the field during June and July of the same year both in the Old and the New Tobacco Belt.

Blue mold was first reported in 1948 on May 17 near Norwich in Oxford County. Three days later it was reported five miles east of Delhi in Norfolk County; and, in the following week, it was found on 10 farms in Norfolk County and at Cottam in the Old Belt. Sporadic occurrences in

both the Old and the New Belt were reported but the disease was not so prevalent as in 1947. Some injury was caused in June and early July to field plants.

The first occurrences of blue mold in 1949 were reported on May 15 at Princeton and two days later at Glen Meyer (Figure 1). It did not appear in the Old Belt, however, until the first week in June; in the meantime it had become widespread in Norfolk County in the New Belt.

In 1950, it was observed on May 11 in the Langton district, Norfolk County, but did not appear in the Old Belt until several weeks later. There was little spread in the seedbeds or injury in the field. The fungus was present during the first part of July on many field plants in the New Belt, and in scattered locations in the Old Belt. In summary, it may be said that only minor injury resulted from the disease in 1948, 1949, and 1950. It may be added that blue mold has not yet appeared in the tobacco-growing areas around Port Hope, east of Toronto, or in the province of Quebec.

#### METEOROLOGICAL CONDITIONS IN RELATION TO INCIDENCE OF BLUE MOLD IN ONTARIO

It is known that meteorological conditions are of primary importance in relation to the incidence and severity of blue mold in any given area. For this reason, an analysis was made of relevant meteorological data collected at the Harrow Station. Daily maximum and minimum temperatures, hours of sunshine, and precipitation for critical months during the 1938 to 1950 period, when blue mold occurred in Ontario, are presented graphically in Figures 2-4.

Tables 3, 4, 5, and 6 contain summaries of relevant weather conditions during the critical weeks prior and subsequent to blue mold infection for the same period of years.

An examination of Figures 2-4 revealed the following relationship between the occurrence of the disease and certain climatic factors. In 1938 (Figure 2), when blue mold first occurred in Ontario, wet cloudy weather prevailed during May. In June, rain fell on 7 of the first 11 days and, during the first two weeks, the mean of the maximum and minimum temperatures were 76° F. and 56° F., respectively. Despite these favourable conditions, the disease did not make its appearance until June 25, near the end of the transplanting season.

In 1939, May was generally warm and dry, with temperatures from 3° and 5° F. above normal. Precipitation occurred only on 6 days during the month. During the first 10 days of June, temperatures were relatively high and rainfall was recorded on 4 days. Rain fell on June 9 and 10, with a temperature decline. An examination of Figure 2 indicates that, in the next 5-day period prior to the disease outbreak on June 20, temperatures were relatively high, with a minimum of 60° F. on June 18. During the same period, rain fell on June 15 and 19. Rain fell also each day from June 21 to 23, notwithstanding more than 8 hours of sunshine daily until June 28. It might also be mentioned that 3 of the 4 occurrences of rainfall during this period happened during the night; thus abundant sunshine during the daytime tended to inhibit sporulation and germination.

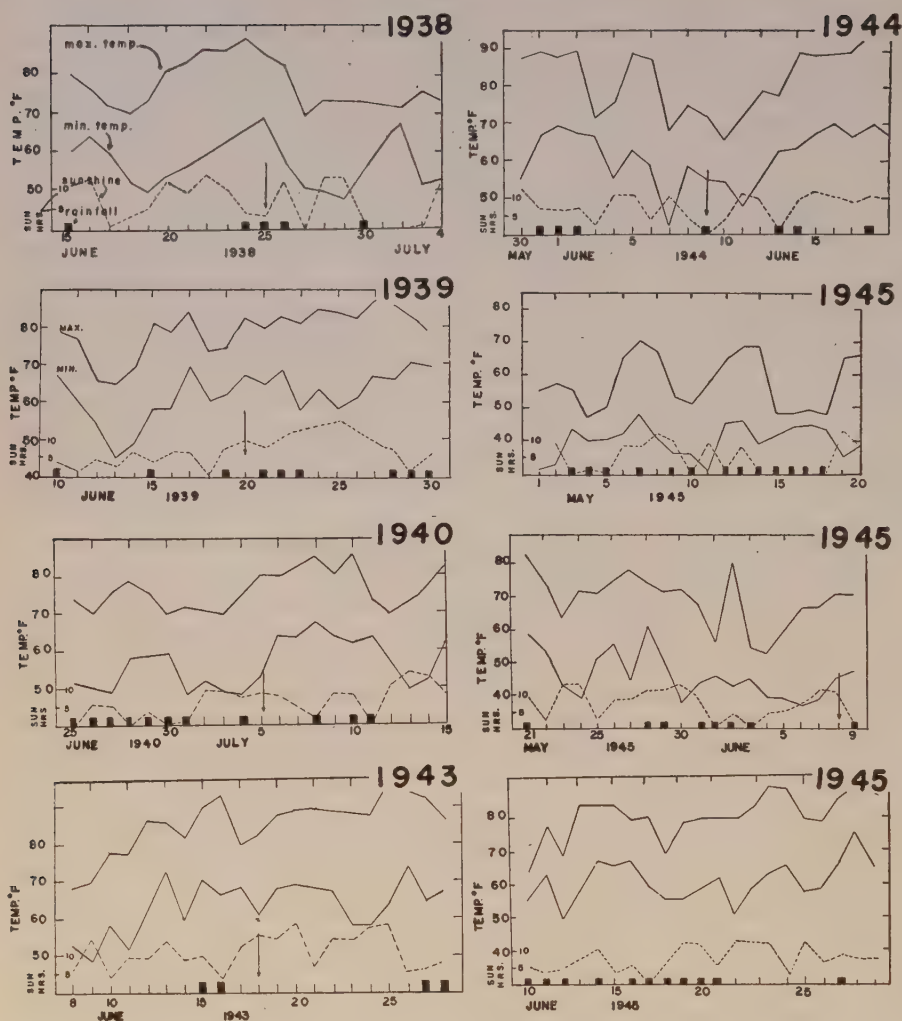


FIGURE 2. Graphical presentation of weather data preceding and subsequent to outbreaks of blue mold in the years from 1938 to July 1945. Blue mold was not present in 1941 and 1942. The vertical arrows indicate the first report of the disease. The daily occurrence and not the amount of rainfall is indicated. From 1945, when the disease was first epidemic, to 1949, the weather data are included for the entire critical months of May, June, and part of July.

In 1940 (Figure 2), precipitation during May was well above normal, with rainfall recorded on 19 days. In June also precipitation was considerably above normal and rain fell on 18 days. Actually, rainfall occurred daily from June 22 to July 1. Temperatures were only slightly above normal throughout June. Notwithstanding the above-mentioned favourable conditions for blue mold, only a single case was recorded, on the relatively late date of July 5.

In 1941, temperatures during May were from 2° to 5° F. above normal. Rainfall occurred on 11 days and was well distributed throughout the month. Sunshine of 5 hours or more was recorded on all but 6 days. In

contrast, precipitation in June was much below normal. During the first half of the month, rain fell on 11 days, but near-drought conditions prevailed for the latter half of the month and well into July. The disease was not reported during this year.

In 1942, precipitation during May was unusually high, with rainfall on 14 days. Temperatures were generally low and sunshine was below average. Rainfall was abundant during the first half of June, but drought conditions prevailed during the latter half. No disease was recorded during this year.

In 1943, rainfall was again excessive during May. Daily rains were recorded from May 2 to 11 and from May 15 to 20. In fact, precipitation occurred on 21 days during the month and temperatures were 2° F. below average. An examination of Figure 2 shows unusually high temperatures accompanied by only 2 occurrences of rainfall in the period preceding the 2 cases of blue mold reported in this year. One week of dry, sunny weather followed the initial outbreak and no spread of the disease occurred.

In 1944, rainfall in May was 25 per cent below normal and temperatures were 6° F. above average. A single case of blue mold was reported on June 9, and an examination of Figure 2 shows that it followed a cool, cloudy period which was preceded by a 3-day rainy period. Temperature and total hours of sunshine rose rapidly subsequent to the outbreak period and no spread of the disease was observed.

In 1945, rainfall occurred almost daily from May 1 to 20 (Figure 2) and was accompanied by relatively low temperatures and cloudy weather. From May 21 until the end of the month, the weather was generally warm, with abundant sunshine. However, rain fell on 6 days between May 28 and June 3 and little sunshine occurred during the latter three days. Five days later, on June 8, blue mold appeared. Then rain again fell on 11 of the next 13 days and during this period blue mold spread rapidly and was soon prevalent throughout the entire tobacco-growing districts bordering Lake Erie. Beginning on July 13 (Figure 3), a field outbreak of the disease occurred in epidemic proportions over the entire area of the New Belt. This outbreak occurred shortly after a rainy period from July 5 and 8 and during a wet period from July 13-15 when the relative humidity was near saturation for 3 days.

In 1946, frequent and persistent rainfall accompanied by low temperatures (Figure 3) preceded the blue mold outbreak reported on May 18. Cloudy, cool weather persisted after the outbreak until May 28. By June 8, the disease was widespread in both tobacco belts. The period from May 31 to June 5 was cool and partly cloudy. The disease spread rapidly from June 10 to 20, and, during this interval, there were frequent showers accompanied by very little sunshine and low temperatures.

In 1947, the first outbreak was recorded on May 11, which was earlier than any previous record in Ontario. This was preceded by a wet period at the beginning of May, accompanied by near-freezing temperatures and very little sunshine (Figure 3). Following the initial outbreak, frequent rainfall and relatively low temperatures prevailed for the remainder of May. During this period, the disease became widespread in all districts.

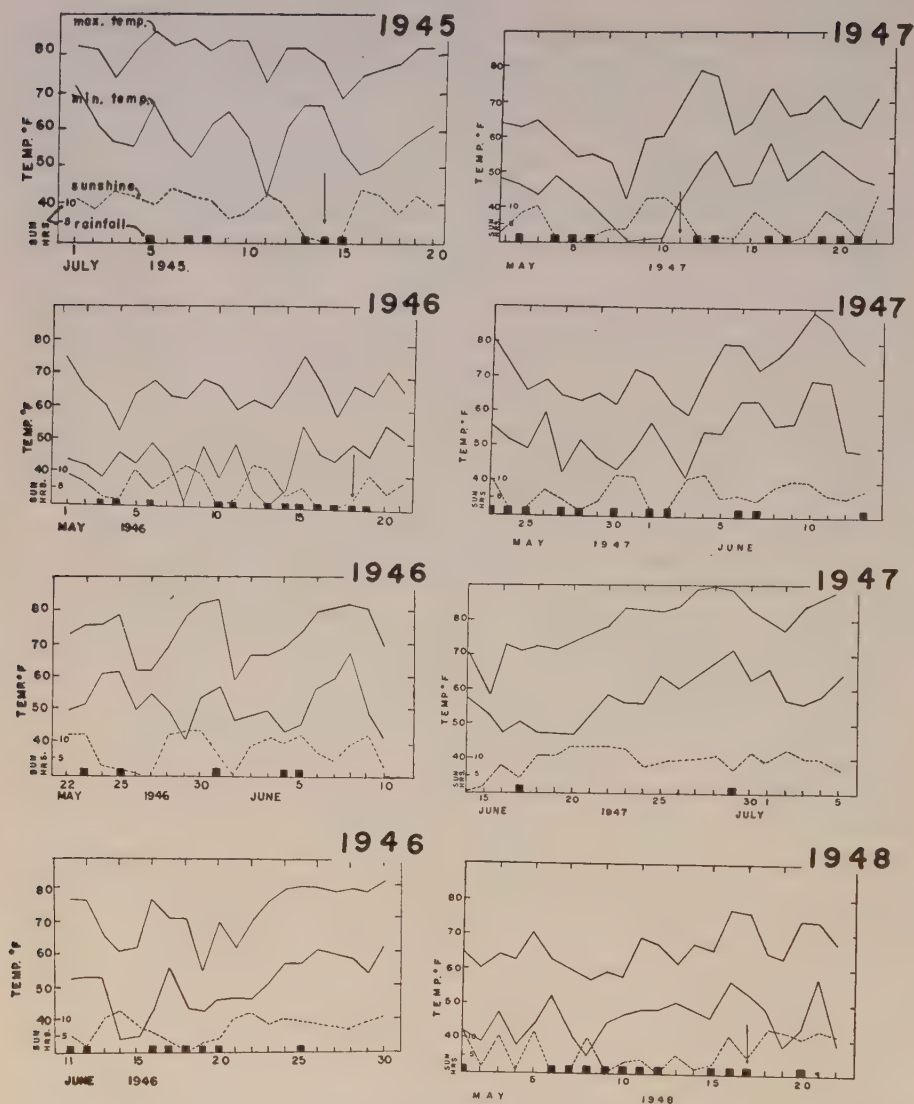


FIGURE 3. Graphical presentation of weather data preceding and subsequent to outbreaks of blue mold in the years from July, 1945, to May, 1948. The vertical arrows indicate the first report of the disease, except the one in July, 1945, which marks a widespread field epidemic.

In 1948, two weeks of frequent rainfall and low temperatures preceded the initial outbreak on May 27 (Figure 3). During the remainder of the month little rainfall was recorded, and abundant sunshine with relatively high temperatures prevailed (Figure 4). Frequent rainfall accompanied by relatively low temperatures prevailed in the period June 4 to 14. Temperatures were relatively high during the latter part of June, but frequent rains during this period favoured widespread field infection.

In 1949, the initial record of blue mold on May 15 was preceded by two weeks of abundant sunshine, widely-fluctuating temperatures, and no

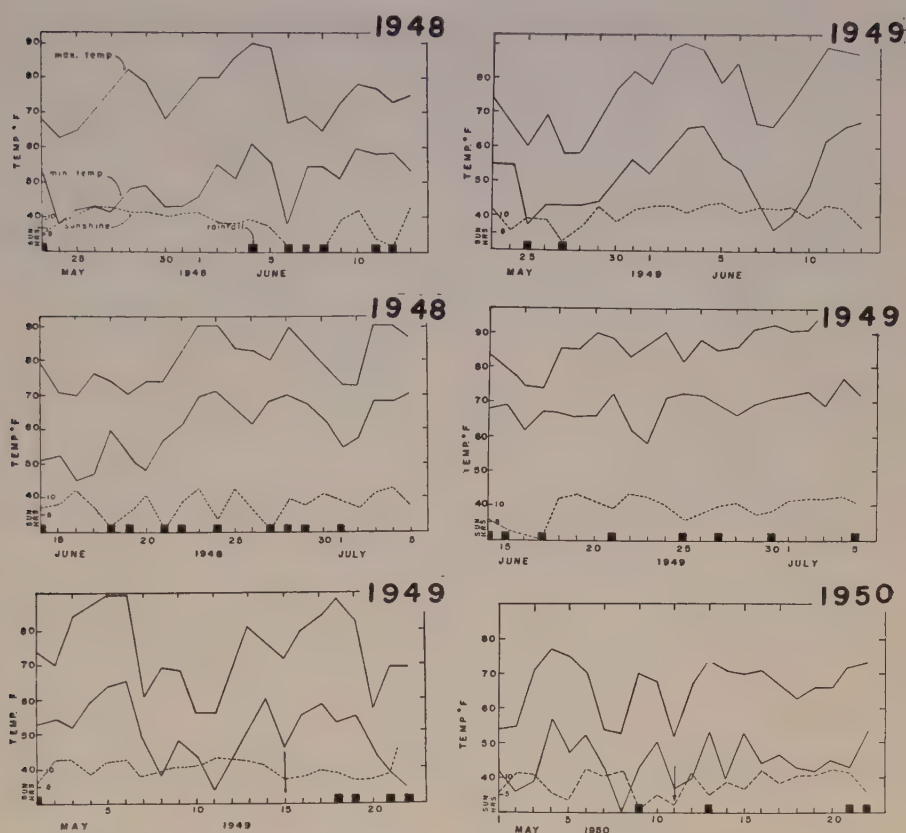


FIGURE 4. Graphical presentation of weather data preceding and subsequent to outbreaks of blue mold in the years from May, 1948, to May, 1950. The vertical arrows indicate the first report of the disease. The daily occurrence and not the amount of rainfall is indicated

rainfall at all (Figure 4). From then until May 27, there was frequent rainfall and temperatures were relatively low. Following this, in the period between May 27 and June 14, temperatures were high, sunshine was abundant, and no rainfall was recorded. Subsequently, the disease was relatively unimportant, though late in the season reports of its occurrence were received from widely-scattered districts.

In 1950, the first record of blue mold was on May 11. Prior to this date, only a single rainfall was recorded, namely, on May 9 (Figure 4). Prevailing temperatures during the period were low and, in fact, dropped below  $35^{\circ}$  F. on May 8. Rainfall was infrequent during the remainder of the month and spread of the disease was negligible. However, during late June and early July, the disease in the field assumed epidemic proportions in certain districts, mainly in the New Belt.

An examination of Tables 3, 4, 5, and 6 further indicates the relationship of certain weather factors to the incidence of the disease. During the years from 1938 to 1944, when blue mold occurred to only a limited extent in Ontario, average temperatures prior to initial outbreaks were approxi-

TABLE 3.—WEATHER CONDITIONS IN ONTARIO DURING PERIODS PRECEDING TOBACCO BLUE MOLD OUTBREAKS FROM 1938 TO 1950

| Year  | Period         | Temperature (° F.) |       |         |       | Hours sun-shine | Relative humidity |      | Days of rain-fall |
|-------|----------------|--------------------|-------|---------|-------|-----------------|-------------------|------|-------------------|
|       |                | Maximum            |       | Minimum |       |                 | A.M.              | P.M. |                   |
|       |                | Mean               | Range | Mean    | Range |                 |                   |      |                   |
| 1938  | June 15-24     | 80.4               | 70-91 | 58.8    | 49-66 | 8.5             | 77                | 64   | 2                 |
| 1939  | June 10-19     | 74.5               | 64-84 | 58.3    | 45-69 | 4.2             | 77                | 64   | 4                 |
| 1940  | June 22-July 4 | 72.7               | 64-78 | 53.7    | 48-63 | 4.4             | 81                | 64   | 11                |
| 1943  | June 6-15      | 77.0               | 64-89 | 58.3    | 48-70 | 7.8             | 84                | 67   | 4                 |
| 1944  | May 30-June 8  | 82.0               | 68-88 | 59.7    | 42-69 | 7.6             | 81                | 64   | 3                 |
| 1945* | June 1-7       | 62.0               | 52-66 | 41.0    | 37-45 | 4.8             | 85                | 76   | 3                 |
| 1946* | May 8-17       | 65.0               | 58-76 | 42.3    | 30-54 | 5.7             | 84                | 76   | 7                 |
| 1947* | May 1-10       | 57.8               | 43-64 | 40.3    | 31-48 | 6.0             | 81                | 73   | 5                 |
| 1948  | May 6-16       | 64.0               | 58-77 | 46.9    | 35-56 | 3.5             | 90                | 79   | 9                 |
| 1949  | May 4-14       | 73.0               | 61-90 | 50.0    | 33-65 | 9.7             | 77                | 53   | 0                 |
| 1950  | May 1-10       | 64.0               | 53-77 | 43.0    | 28-57 | 7.8             | 70                | 54   | 1                 |

\* Disease epidemic.

TABLE 4.—WEATHER CONDITIONS IN ONTARIO DURING PERIODS FOLLOWING TOBACCO BLUE MOLD OUTBREAKS FROM 1938 TO 1950

| Year  | Period         | Temperature (° F.) |       |         |       | Hours sun-shine | Relative humidity |      | Days of rain-fall |
|-------|----------------|--------------------|-------|---------|-------|-----------------|-------------------|------|-------------------|
|       |                | Maximum            |       | Minimum |       |                 | A.M.              | P.M. |                   |
|       |                | Mean               | Range | Mean    | Range |                 |                   |      |                   |
| 1938  | June 25-July 2 | 75.4               | 69-85 | 57.9    | 50-68 | 5.6             | 76                | 64   | 2                 |
| 1939  | June 20-30     | 82.5               | 78-86 | 64.3    | 58-70 | 9.6             | 76                | 64   | 6                 |
| 1940  | July 5-15      | 78.9               | 72-85 | 59.6    | 49-67 | 7.8             | 79                | 68   | 4                 |
| 1943  | June 16-26     | 88.2               | 79-96 | 65.3    | 58-74 | 11.3            | 81                | 22   | 1                 |
| 1944  | June 9-16      | 79.1               | 65-89 | 59.4    | 47-70 | 7.0             | 88                | 62   | 3                 |
| 1945* | June 8-21      | 76.0               | 63-83 | 57.7    | 45-67 | 6.2             | 92                | 72   | 11                |
| 1946* | May 18-27      | 69.0               | 61-75 | 53.9    | 46-61 | 5.6             | 91                | 82   | 4                 |
| 1947* | May 12-22      | 69.3               | 61-79 | 51.4    | 46-59 | 4.1             | 81                | 77   | 7                 |
| 1948  | May 18-27      | 68.5               | 63-76 | 44.2    | 37-57 | 10.7            | 79                | 53   | 1                 |
| 1949  | May 16-25      | 73.2               | 60-89 | 50.7    | 38-58 | 7.4             | 85                | 66   | 5                 |
| 1950  | May 12-24      | 70.4               | 63-80 | 40.2    | 40-53 | 9.5             | 80                | 58   | 4                 |

\* Disease epidemic

TABLE 5.—AVERAGE TEMPERATURES PRECEDING TOBACCO BLUE MOLD OUTBREAKS DURING PERIODS OF MILD, MODERATE, AND SEVERE DISEASE\*

| Period    | Disease severity | Mean temperatures (° F.) |         |         |         |
|-----------|------------------|--------------------------|---------|---------|---------|
|           |                  | Maximum                  |         | Minimum |         |
|           |                  | Range                    | Average | Range   | Average |
| 1938-1944 | Mild             | 73-80                    | 77.3    | 54-60   | 57.7    |
| 1945-1947 | Severe           | 58-65                    | 61.6    | 40-42   | 41.6    |
| 1948-1950 | Moderate         | 64-73                    | 67.0    | 47-50   | 46.6    |

\* From columns 1 and 3 of Table 3.

TABLE 6.—AVERAGE TEMPERATURES FOLLOWING BLUE MOLD OUTBREAKS DURING PERIODS OF MILD, MODERATE, AND SEVERE DISEASE\*

| Period    | Disease severity | Mean temperatures (° F.) |         |         |         |
|-----------|------------------|--------------------------|---------|---------|---------|
|           |                  | Maximum                  |         | Minimum |         |
|           |                  | Range                    | Average | Range   | Average |
| 1938-1944 | Mild             | 75-88                    | 80.7    | 58-65   | 61.3    |
| 1945-1947 | Severe           | 69-76                    | 72.5    | 51-58   | 54.3    |
| 1948-1950 | Moderate         | 69-73                    | 70.8    | 44-51   | 47.1    |

\* From columns 1 and 3 of Table 4.

mately 16° higher than at the corresponding time in the epidemic period of 1945 to 1947. Average temperatures following initial outbreaks from 1938 to 1944 were also approximately 8° higher than in the epidemic period of 1945 to 1947. The main reason for this great difference in temperature, of course, lies in the fact that, during the years from 1938 to 1944, the disease at no time appeared earlier than June 9 and, in fact, made its initial appearance as late as July 5, whereas, in the epiphytotic period, it appeared as early as May 11 and no later than June 8. In the period 1948 to 1950, when the disease was less severe than in the 1945 to 1947 period though, nevertheless, widespread, average maximum and minimum temperatures preceding initial outbreaks were approximately 10° lower than for the 1938-1944 period. As for periods subsequent to initial outbreaks, average maximum and minimum temperatures were approximately 10° to 14° lower, respectively, than for the 1938-1944 period. This is again explained by the fact that from 1948 to 1950 the disease made its initial appearance unusually early in the season, e.g., May 11-17. It should also be noted that, in the 1938-1944 period, average maximum temperatures during the period immediately following initial outbreaks were 80.7° F., obviously a critical temperature and one which undoubtedly proved limiting to the disease throughout this period.

As far as hours of sunshine are concerned, it may be noted that prior to initial outbreaks of disease daily sunshine varied from 4.8 to 6.0 hours during the epidemic years of 1945 to 1947, whereas during the years 1938 to 1944, when disease outbreaks were mild, it varied from 4.2 to 8.5 hours, and from 3.5 to 9.7 hours during 1948 to 1950. In periods following

initial outbreaks, hours of sunshine varied from 4.1 to 6.2 hours during the epidemic period of 1945 to 1947, and from 5.6 to 11.3 hours during periods of light or moderate disease.

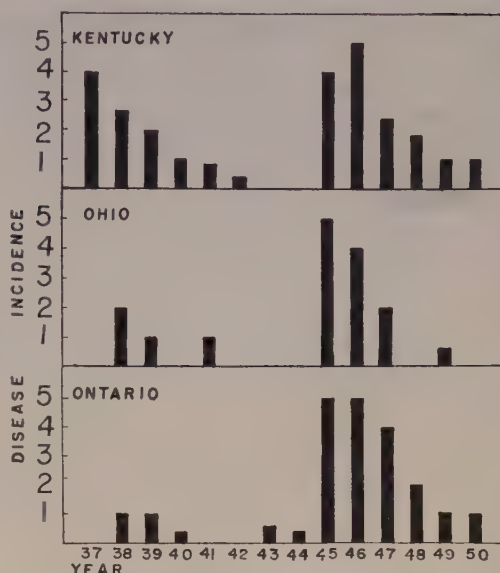
The relative humidity, generally speaking, was highest during the epidemic period of 1945 to 1947, both before and after initial outbreaks. Prior to initial outbreaks, it varied from 73 to 85 per cent during 1945 to 1947, from 64 to 84 per cent from 1938 to 1944, and from 54 to 90 per cent during the 1948 to 1950 period. Following initial outbreaks, relative humidity within the epidemic period 1945 to 1947 varied from 77 to 92 per cent, whereas it ranged from 62 to 88 per cent in the period 1938 to 1944, and from 66 to 85 per cent in the years 1948 to 1950.

### DISCUSSION

It is evident from the meteorological data studied that low temperatures, high humidity, numerous showers, and reduced sunshine, all contributed towards optimum environmental conditions for the rapid development and spread of blue mold in Ontario in the years 1945 and 1946. Probably a low inoculum potential during the early seedbed season may have proved to be more of a limiting factor than weather conditions in disease development during the period from 1948 to 1950. Two sources of blue mold inoculum are recognized as being involved in disease outbreaks in Ontario, namely, wind-borne spores and overwintering of the fungus. If overwintering sources are present, they are probably very much reduced in number because of the general practice of steaming old beds. Outbreaks entirely dependent on overwintering of the fungus would require several weeks of favourable conditions for the inoculum from primary and early secondary outbreaks in seedbeds to reach a potential sufficient to initiate a general epidemic. However, a heavy spore shower from the south, followed by conditions favourable for infection and development of the fungus lasting for a period of from one to two weeks, could result in a general outbreak. It is probable, therefore, that the decline of blue mold in Ontario following the 1945-1947 period has been due more likely to a lack of sufficient inoculum in the form of heavy spore showers, such as occurred in 1945 and 1946, than to weather conditions. Also, the fact that protectant fungicides have been generally and widely used after 1946 would prevent a rapid build-up of inoculum and hence cause a reduction in disease incidence. There has been a tendency on the part of growers, however, to omit spraying after light disease years, and, when the disease has been absent or mild for several years, many seedbeds are left unprotected. Therefore, if a heavy spore shower does occur after several light disease years, it is likely to result in a large number of seedbeds becoming simultaneously infected.

The interaction of temperature, moisture, and sunshine also plays a part. The adverse effect of high temperature may be counteracted by high humidity, or by overcast sky and drizzling rain, as evidenced by field outbreaks of the disease during periods of relatively high temperature when humidity is high, or by abundant rainfall, or by lack of sunshine (Figure 3, 1945). Abundant sunshine and dry weather hinder disease development, even though temperatures may be optimum. It is known that temperature is one of the most important factors involved in outbreaks of blue mold of tobacco. During the 1938-1945 period, when initial occurrences of blue

FIGURE 5. Relative incidence of blue mold in Kentucky and Ohio from 1937 to 1950 and its relationship to the incidence of the disease in Ontario. Disease incidence: 1, light; 2, moderate; 3, severe; 4, very severe; 5, epidemic.



mold in Ontario were always relatively late, temperatures undoubtedly proved limiting on many occasions when other factors proved favourable for development and spread (Table 6). On the other hand, during the 1945-1950 period, temperatures following initial disease outbreaks would not appear to have retarded disease development (Tables 5, 6). However, the relation of low temperatures to the viability of air-borne sporangia and their dissemination over great distances prior to disease outbreaks should be emphasized. Of course, sunshine and rainfall may be equally important factors in the long range dispersal of viable sporangia. In any case, it would appear that factors other than temperature have limited the incidence of blue mold in Ontario since 1947. Lastly, attention should be drawn to the fact that, regardless of how favourable weather conditions may be, the incidence and severity of primary outbreaks of blue mold are dependent upon the abundance of inoculum available. It appears that the most suitable conditions for a blue mold epidemic in Ontario are as follows: Heavy spore showers from the south during May or the first week in June; temperatures ranging from a maximum of 75° F. to a minimum of 50° F.; moisture conditions that prevent the drying of the leaf surface of seedlings. Frequent showers, and high humidity in the absence of sunshine, are necessary to maintain a moist seedbed environment, and thus to favour infection and sporulation. These conditions, when present, alleviate to some extent the adverse effects of high temperatures. During the initial outbreak periods in Ontario since 1945, high temperatures do not appear to have been a limiting factor in disease development.

Three major periods which merit detailed consideration are apparent in the epidemiology of the blue mold disease of tobacco in Ontario from 1938 to 1950. In the first period, from 1938 to 1944, the disease was confined to a few farms in the Old Tobacco Belt (Table 2). In 1938, 1939, and 1940, blue mold did not appear until late in June. In 1941 and 1942, the disease was absent, and in 1944 only a single case was reported.

A study of disease occurrence in the United States (Table 1) suggests the likely sources of inoculum in the 1938 and certain other outbreaks in southern Ontario. In 1932 and 1933, which were epiphytotic years in the United States, the disease spread as far north as Lancaster County, Pennsylvania. After 1933, blue mold decreased in severity, and, in 1936, a light disease year, it spread to Kentucky. In 1937, the disease was severe in Kentucky and present in Indiana. However, it was not until 1938 that blue mold made its appearance in Ohio, and also in Ontario. The tobacco-growing areas in Ohio are only 125 miles southwest of Essex County, Ontario (Figure 6). Therefore, it is highly probable that wind-blown spores from Ohio or Kentucky served as the source of inoculum for the initial 1938 outbreak of blue mold in Ontario. The dissemination of *P. tabacina*, and the rapid spread of the disease in the United States, are attributed to air currents. The duration of southerly winds in Ontario is of sufficient frequency during May, June, and July to carry the spores of *P. tabacina* from the Ohio-Kentucky area to Ontario. The average percentage of recorded wind directions for the ten-day period preceding\* disease outbreaks are as follows: 1938 to 1946, S-13 per cent, SW-32 per cent, SE-20 per cent, others, 35 per cent; 1945, S-5 per cent, SW-45 per cent, SE-20 per cent, others 30 per cent; 1946, S-0 per cent, SW-25 per cent, SE-35 per cent, others, 40 per cent. It is evident, therefore, that southerly and particularly southwesterly winds were predominant preceding the disease outbreaks.

Newhall (14) trapped viable conidia of *Peronospora destructor* at elevations up to 1500 feet and he states that spores could have been found above 1500 feet if sufficient search had been made. Pinckard (15) noted that, if atmospheric saturation persists, the mature sporangia of *P. tabacina* remain attached to the sporangiophore. He concluded that, during periods of continuous rain or during periods with no vapour-pressure deficit in the vicinity of the sporangia, little dissemination occurs.

Although fungus spores are carried by the wind for variable distances, as far as the causal organism of blue mold is concerned little is known about the relationship between spore viability and their capacity for travel. As previously stated, sporulation of *P. tabacina* is most abundant at dawn, or shortly after, and most of the spores are dead by 11.00 a.m. (6). Furthermore, the conidia are sensitive to high temperatures and sunlight. However, a few spores have been found to remain viable under favourable conditions for two weeks and longer (4, 25). Weather conditions in Ontario preceding the 1938 and 1939 disease outbreaks were unfavourable for the maintenance of viable conidia (Figure 2). Therefore, in 1938 and 1939, it is likely that relatively few viable spores reached Essex and Kent counties. It is possible that during a prolonged period of cool, intermittently wet weather, with little sunshine, a certain number of the spores would remain viable long enough to be carried to districts 150 or 200 miles distant. Even then, chances are not great for viable spores in sufficient numbers to reach infection courts under environmental conditions favourable for incubation, sporulation, and dissemination. Incidentally, it might be mentioned that the capacity for spore production among the downy mildew fungi is great.

\* Records were taken for ten days preceding a five-day period allowed for incubation prior to the first report of the disease.

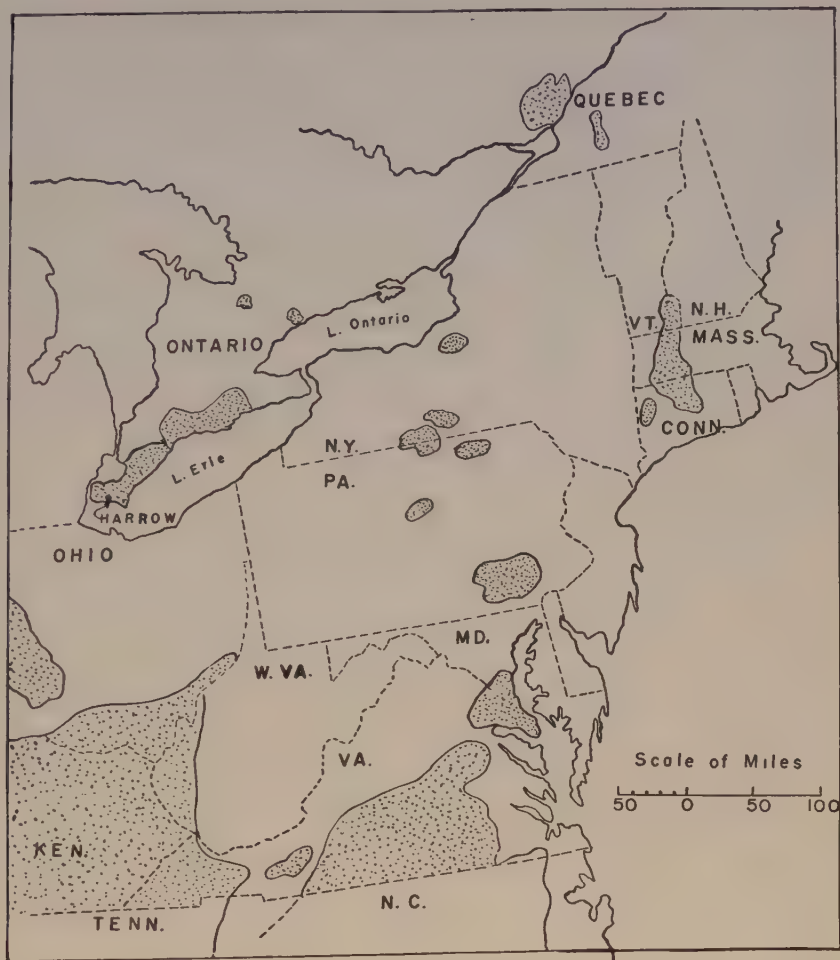


FIGURE 6. Tobacco-growing areas in the United States and their geographic relation to those in Canada. Note the proximity of the Ohio-Kentucky area to southern Ontario, and the Connecticut Valley to southern Quebec.

For instance, it has been reported that *Peronospora schleideni*, a close relative of *P. tabacina*, may produce 140,000 conidia per square inch of leaf surface.

The localization of disease outbreaks during the period from 1938 to 1944 may be explained on the basis of only partially-favourable weather conditions, a lack of heavy spore showers, and the late seasonal appearance of the disease. During its first three years in Ontario, blue mold did not occur until near the end of the plant-bed season. By this time temperatures were high, general weather conditions for maximum sporulation were no longer optimum, and seedlings were for the most part beyond their most susceptible stage. However, it should be noted that weather conditions were highly favourable for blue mold development in Ontario during the latter half of June, 1940 (Figure 2). In this year, however, disease incidence was very low in the Kentucky-Ohio area and this condition would likely

reduce or eliminate the major source of wind-borne inoculum for infection in southern Ontario. If Ontario regions had experienced a spore shower in 1940, there is little doubt that an epidemic would have developed here as occurred in Pennsylvania the same year. In 1943 and 1944, high temperatures, abundant sunshine, and low precipitation during the outbreak periods served to limit secondary infections.

The fact that until 1945 the fungus did not appear in the concentrated flue-cured tobacco area of Ontario lends further support to the hypothesis that the Kentucky-Ohio area serves as the major source of inoculum for Ontario. Lancaster County, Pennsylvania, is only 250 miles southwest of the nearest tobacco-growing area in Ontario (Figure 6). Judging from the geographic location of Pennsylvania, it is evident that, if wind-blown spores were originating from this area, infection in the highly-concentrated tobacco areas of the New Belt (Figure 1) would have been observed at an earlier date. Also, it should be noted that a single case of blue mold was present in Ontario in 1940 when Pennsylvania experienced an epidemic. It would appear, therefore, that the initial source of inoculum for the 1938 and 1939 outbreaks of blue mold in Ontario was from the Ohio-Kentucky area. However, in the period 1940 to 1944, overwintering of the fungus in Ontario was the probable source of inoculum.

Although it is highly probable that the source of the initial inoculum in 1938 was the Kentucky-Ohio area, it may be noted that the disease was either absent or very mild in Ohio and Kentucky in 1943 and 1944 (Table 1 and Figure 5). In 1943 and 1944, single cases were reported from Essex County (Table 2). In order for the inoculum initiating these outbreaks to have been air-borne its point of origin would have been no closer than Virginia, which is unlikely, since the disease was absent in Kentucky in 1944. Thus, overwintering would appear to be the logical source of inoculum in Ontario during 1943 and 1944 even though the disease was not reported here for the two previous years. In 1943 and 1944, the disease occurred in the same areas as in 1938 and 1939, but not on the same farms. This fact could be explained by the possibility that the fungus had overwintered for several years in infected beds without having been reported, and subsequent infection and dissemination occurred. In this connection, it may be mentioned that the single case of the disease in 1940 occurred in a seedbed that had been infected in 1938. It may also be possible that the oospores of this fungus are capable of remaining viable for two years or longer. Since the disease was absent in Ohio in 1940, and appeared late in the same season in Kentucky, it is unlikely that a sufficient number of viable spores reached Ontario to initiate a single infection in one of the eight seedbeds infected in 1938.

The decline and disappearance of the disease in the Kentucky-Ohio inoculum reservoir during the period from 1939 to 1944 eliminated this, the nearest outside, source of inoculum. As previously indicated, the occurrence of the disease in the Old Belt during this period strongly suggests that overwintering can and does occur in Ontario. However, the consistently-delayed appearance of the disease from 1939 to 1944, the absence of the disease in seedbeds infected in the previous year, and the failure to find oospores, are opposed to the overwintering hypothesis. Even if oospores were present in some of the diseased tissue, evidence that their development

has led to primary infections in Ontario is still wanting. Also, the widespread practice of steaming would tend greatly to reduce the chances of overwintering from these sources. However, the practice of steaming hotbeds is not so general in the Old Belt, where flat, cotton or sash-covered seedbeds are mostly used, whereas in the New Belt greenhouses are used for production of seedlings. Further evidence in support of overwintering will be given in the discussion covering the second period of blue mold occurrence in Ontario.

The second blue mold period in Ontario comprises the epidemic years 1945, 1946, and 1947. In 1945 and 1946, a general outbreak of the disease occurred throughout the entire Old and New Tobacco Belts. The almost simultaneous occurrence of the disease over widely-scattered areas in both belts in 1945 and 1946 leaves no doubt that the infections resulted from spore showers. This conclusion was even more evident from the fact that Kentucky, Ohio, and Pennsylvania simultaneously experienced epidemics (Table 1, and Figure 5). In 1945, Ohio suffered its most severe and widespread outbreak of the disease up to that time; as did Kentucky in 1946. The epiphytotics in these states, no doubt, provided a large reservoir of inoculum for the spore showers that occurred in southern Ontario. The 1945 spore shower in Ontario occurred during a period when optimum weather favoured the development of the disease (Figure 3). In addition to optimum weather conditions and abundant inoculum, most of the seedbeds in that year were unsprayed. It is known that intermittent rains accompanied by extended periods of high humidity favour sporulation, but inhibit dissemination (15). Therefore, the weather conditions preceding the 1945 outbreak, during which no rain fell for 4 or 5 days but cloudy weather prevailed, undoubtedly favoured long distance dissemination of spores. An influx of wind-borne spores would account for the simultaneous appearance of primary infections in large areas of both the Old and the New Belts in 1945. In 1946, however, one week of daily rains preceded the initial outbreaks, a fact that would account for the fewer and more scattered reports of primary infections in 1946. Nevertheless, the alternately clear and cloudy days, the latter usually accompanied by rain, that followed the 1946 outbreak favoured rapid dissemination. This is in contrast to the ten days of wet weather that followed the 1945 outbreak (Figure 3).

In 1946, the first report of the disease was on May 18 and in subsequent years the disease has always appeared near the middle of May. This contrasts with its appearance in late June or early July during the 1938 to 1944 period.

Another general outbreak of blue mold occurred in 1947, though the resultant injury was considerably less than in the previous two years. The reduced injury can be attributed largely to the widespread and effective use of the fungicide Ferbam. Weather conditions during and following the 1947 outbreak were favourable for disease development until June 10, after which dry, sunny weather prevailed (Figure 3).

The years 1948 to 1950 may be considered as the third period in the history of blue mold in Ontario to date. These were light disease years following the epiphytotics of 1945, 1946 and 1947. In 1948, blue mold was less widespread than in 1947, although sporadic infections occurred in most areas in both the Old and the New Tobacco Belts. The outbreak period

was followed by dry, sunny weather (Figures 3, 4), and little general spread of the disease occurred. Much of the spread occurred in the field during July, when cloudy, rainy weather prevailed. Ferbam was again widely used as a protectant fungicide.

The disease continued to decline in importance in 1949 and 1950. However, enough seedbeds were infected by the end of the plant-bed season during both years to furnish inoculum for widespread field outbreaks in July. Nevertheless, the lower leaves of the field plants proved to be more commonly attacked than the middle leaves and severe injury was observed in only a few fields.

The source of inoculum for the initial outbreaks in the period 1947 to 1950 is more difficult to determine than that in 1945 and 1946 which definitely was wind-borne spores. In 1947, the disease was general in Ohio and Kentucky and, therefore, an inoculum source for light spore showers in the Old Belt of Ontario was available. However, the earliest reports of the disease in Kentucky were on May 8, in Ohio on May 17, and in Ontario (Leamington) on May 11 (Table 1). It is unlikely, therefore, that wind-borne spores from Kentucky or Ohio served as a source of inoculum for the initial outbreak at Leamington in 1947. Also, because of general and severe infections in 1946, the probability of overwintering would be greater and a larger number of overwintering sources would have been present to initiate primary infections in 1947. The possibility of spore showers from the south occurring some time after the initial 1947 outbreak cannot be eliminated. The disease did not appear in the New Belt, however, until two weeks after its first appearance in the Old Belt. Therefore, inoculum for the New Belt infections may have originated from primary and secondary outbreaks in the Old Belt.

In 1948, the disease was not reported in Ohio, and was light in Kentucky. Therefore, it is unlikely that spore showers originating from these areas occurred in Ontario in 1948. Also, in 1948, 1949, and 1950, blue mold in Ontario was reported first in the New Belt, and the disease did not appear in the Old Belt until from one to three weeks later. This suggests the absence of spore showers preceding the initial outbreaks. When spore showers occur in Ontario, they may be expected to occur in both the Old and the New Belts because of the proximity of the former area to the inoculum source in Kentucky. Furthermore, the circumstances in connection with the initial occurrence of blue mold in 1948 strongly indicated that the source was overwintering of the causal fungus for the following reasons. Firstly, it occurred in a seedbed known to be severely infected in the previous year. Secondly, the presence of weeds in parts of the seedbed indicated an imperfect steaming job. Thirdly, the original infection comprised only a few plants, the leaves of which were less than one inch wide and were in direct contact with the soil. Finally, seedbeds in the immediate vicinity remained free from the disease until approximately one week later.

In 1949, the Ohio and Kentucky tobacco-growing areas experienced only light attacks of blue mold. The disease outbreak in the Old Belt of Ontario, which is nearest to the Kentucky-Ohio area, did not occur until several weeks after the initial outbreaks in the New Belt. It is unlikely, therefore, that spore showers from the south occurred in the Old or the

New Belt of Ontario in 1949. If spore showers did occur then, it is improbable that seedbeds in the New Belt would be infected prior to those in the Old Belt. In 1950, blue mold was not reported in Ohio and, as in the previous year, the first outbreak in Ontario occurred in the New Belt. From the circumstantial evidence outlined above, it appears probable that oospores rather than spore showers from the Kentucky-Ohio area served as the sources of inoculum for the initial disease outbreak in the period 1947 to 1950.

Up to the present the tobacco-growing areas in Quebec have been free from blue mold. In this regard, it is interesting to note that, when Ohio, Pennsylvania, Kentucky, and Ontario tobacco areas were experiencing severe epiphytotics in 1945 and 1946, the Connecticut Valley, including Massachusetts, experienced only a light general infection. The tobacco-growing areas of southern Quebec (Yasmaska River Valley) are within 150 miles of the northern Massachusetts areas (Figure 6), a distance about equal to that between Ohio and Ontario. It might be predicted that in the event of a severe epidemic occurring in Massachusetts and the Connecticut Valley, with weather conditions prevailing in Quebec similar to those in Ontario during 1940, 1945 or 1946, an outbreak of blue mold would likely occur in the Quebec tobacco-growing areas.

#### THE WARNING SERVICE IN RELATION TO CONTROL MEASURES

It is evident from the data already presented that severe outbreaks of blue mold in Ontario are dependent upon spore showers originating in the Kentucky-Ohio tobacco areas. It should also be noted that the heavy spore showers occurring in Ontario during 1945 and 1946 were closely associated with severe outbreaks in the Kentucky-Ohio areas (Figure 5). It is reasonable to conclude, therefore, that spore showers from Kentucky or Ohio will only occur in sufficient abundance to initiate rapid and general seedbed infections in Ontario during years of severe disease outbreaks in Kentucky. Because of the low incidence of overwintering of the causal organism in Ontario, it seems unlikely that a sufficiently high inoculum potential would obtain from this source to initiate an epidemic in Ontario. The slow build-up of inoculum resulting from the relatively few annual occurrences of overwintering of the causal fungus in Ontario, coupled with the ample time that such outbreaks allow to fully protect seedbeds in surrounding areas by means of sprays or dusts, tend to suggest that under such circumstances control of blue mold would be a relatively simple matter. However, unexpected and widespread spore showers, such as those which occurred in 1945 and 1946, usually result in general outbreaks before adequate general control measures can possibly be implemented and suggest that a complete routine spraying or dusting programme is the only reliable means of control. It is against these "surprise" spore showers that the Ontario tobacco grower must always be on guard, in order to prevent widespread seedbed destruction, such as occurred in 1945 and 1946. Nevertheless, there is arising a tendency on the part of growers to delay or neglect altogether the use of protective measures.

In Ontario, the critical period for severe blue mold infection is from May 7 to June 15. Transplanting is usually well started by June 10, and completed by June 30. In order to have continuous protection against

blue mold infection, it is necessary to apply sprays every seven to ten days during this critical period. If 100 square feet of seedbed is provided for each planted acre of tobacco, there would be more than 10,000,000 square feet of tobacco seedbed in Ontario. At the minimum dosage of  $1\frac{1}{2}$  lb. Ferbam per 40 gal. water to cover 2400 square feet of seedbed, more than 6000 lb. Ferbam per application would be required. On the basis of 5 applications per season at a cost of 80 cents per lb., this material would represent a total annual cost of \$24,000. Much expenditure of labour and funds could be avoided if the incidence of blue mold could be more or less accurately predicted. Since severe disease outbreaks are dependent on spore showers, it is necessary to predict at what time the latter are likely to occur. As stated previously, many growers neglect to spray or dust after a light disease year and, in general, they plan spray schedules on the basis of their previous year's experience with the disease. Following several consecutive years of mild disease, large numbers of unprotected seedbeds remain vulnerable to infection from spore showers from any source. Farmers are often reluctant to spray when the disease has been light or absent for many years, unless they are advised to do so.

In order to predict the occurrence of spore showers in Ontario, it would be necessary to know the distribution and severity of the disease in Kentucky and then evaluate the chances of a spore shower occurring on a basis of wind direction and velocity. It would be necessary also to evaluate the role of weather on spore viability, infection and subsequent disease development. In Table 1, it was noted that blue mold commonly appears in Kentucky during the last week of April and the first week of May. Outbreaks of this disease in Kentucky originate from two sources, namely, overwintering (1939, 1946) and spore showers from the southeast (1938, 1945). In Ontario, outbreaks occur from the middle of May to the first week in June. The interval between outbreaks in Kentucky and those in Ontario is approximately two or three weeks. If during this period complete information regarding the severity and build-up of the disease in Kentucky were available in Ontario, it would be highly possible to put into effect a useful warning system. Such a system would be based on probability of spore showers occurring and on the current local weather conditions. The only alternative would be to maintain a minimum regular spraying or dusting schedule each year during the critical growing period, and to augment this schedule when weather conditions and local outbreaks indicate that increased protection is advisable. Large areas of completely unprotected seedbeds during a year of heavy spore showers and optimum weather conditions could result in a disastrous shortage of seedlings in Ontario.

### SUMMARY

1. Previous work concerning the relation of important climatic and other factors to the incidence and severity of blue mold of tobacco in the various tobacco-growing areas of the United States and Canada is reviewed in some detail.

2. A careful study was made of relevant meteorological data in southern Ontario prior and subsequent to outbreaks of blue mold beginning in 1938, when the disease first occurred in Ontario. During the critical periods of

the epiphytotic years 1945 to 1947, inclusive, average temperatures were considerably lower, total sunshine was less, and relative humidity considerably higher than in other years when the disease occurred to a lesser extent. In years when the disease was epidemic, it invariably appeared earlier in the season than in the other years.

3. Evidence is submitted to indicate that wind-borne inoculum (conidia) in mass quantity, probably originating in Kentucky and Ohio, was likely responsible for the severe outbreaks of blue mold in Ontario, at least in 1945 and 1946. Evidence is also presented that the blue mold organism can and does overwinter under Ontario conditions.

4. It is concluded that mass quantities of wind-borne inoculum from the United States tobacco-growing areas occurring in conjunction with periods of relatively low temperatures, reduced sunshine, prolonged rainfall, and relatively high humidity in Ontario tobacco-growing districts during critical stages of seedling growth, are the circumstances most likely to result in epiphytotics of blue mold in that province.

5. Because of the various circumstances involved it is also concluded that an evaluation of disease incidence in the Kentucky-Ohio tobacco-growing areas in relation to the relevant factors in Ontario during the critical period each season should make possible the issuing of useful warnings to Ontario growers regarding timely and appropriate control measures.

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# THE INHERITANCE OF FLESH COLOUR AND CLUBROOT REACTION IN SWEDES

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## INTRODUCTION

Clubroot, *Plasmodiophora brassicae* Wor., also known as finger and toe disease, clubbing and club-foot in English speaking countries, is destructive to wild and cultivated crucifers in most temperate climates.

The swede crop, *Brassica campestris* var. *napobrassica* DC. is of considerable importance in the Maritime Provinces of Canada, both as a live-stock food and as human food. Although considerable progress has been made towards the development of varieties of swedes which show high resistance to clubroot, little is known of the manner in which this resistance is inherited. Such knowledge would be of considerable value to the plant breeder and plant pathologist.

The research reported herein was conducted at the Experimental Farm, Nappan, Nova Scotia. All findings and comments refer to the swede crop and, in particular, to the varieties used in the experiment with special reference to conditions existing at Nappan, N.S.

The purpose of the investigation was threefold: (1) to study the genetics of host reaction to clubroot; (2) to study the inheritance of flesh colour; (3) to determine if linkage relationships existed between flesh colour and clubroot reaction.

## LITERATURE REVIEW

Davey (2) reports that, in swedes, ratios of 15 : 1 and 3 : 1 for white: yellow flesh colour have been obtained at the Scottish Plant Breeding Station. He also notes that bright yellow and dull yellow flower colour are either controlled by the same genetic factors or are closely linked with factors for white and yellow flesh colour, respectively, in swedes and turnips.

Anderson (1) noted that, in England, white fleshed turnips were more subject to clubroot than were yellow fleshed ones. Rostrup (6) found that swedes were more resistant to clubroot than were turnips.

## MATERIALS AND METHODS

Three highly clubroot resistant S<sub>4</sub> plants of the white fleshed variety Danish Giant were chosen as one group of parents to represent one group of characters. These were designated as plants A, B and C.

Plants D, E and F, the other group of parents, were selected at random from an open-pollinated crop of the yellow fleshed variety Ditmars Bronze Top which was grown on disease-free soil. This variety is known to be highly susceptible to clubroot.

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In 1947 the reaction of the nine possible  $F_1$  hybrids between these two parental groups was studied in the clubroot garden with the progeny of the selfed parents. The performance of the parental progenies gave an indication of the homozygosity of the parents for factors determining clubroot reaction and it was decided to retain only that material involving parents A, C, E and F for further study. Two non-infected  $F_1$  plants from each  $F_1$  hybrid were selfed in the greenhouse during the winter in order to obtain  $F_2$  seed. One of these  $F_1$  plants from each cross was also used in the production of backcross populations. Those  $F_1$  plants used in backcrosses were designated with the subscript (b).

In 1948 two tests of ten replications each were seeded in simple randomized blocks. One test was conducted in the clubroot garden and the other on disease-free soil. The purpose of the latter test was to obtain larger populations for flesh colour studies and to provide a source for random selections from the segregating generations for  $F_3$  progeny trials.

Each test consisted of the following:

1. Progeny of the four selfed parents—A(s1), C(s1), E(s1) and F(s1).
2. Four  $F_1$  hybrid populations—A  $\times$  E, A  $\times$  F, C  $\times$  E and C  $\times$  F.
3. Eight  $F_2$  populations—one from each of two non-infected roots of each  $F_1$  hybrid.
4. Eight backcross populations—produced by crossing a plant of each  $F_1$  hybrid to a plant of its selfed parental progenies.
5. Progeny of the selfed recurrent parent plants—A(s2) and F(s2).

It was not possible to obtain selfed seed from C(s1) and E(s1) plants.

Two-row plots were grown of the  $F_2$  populations while all other generations consisted of one-row plots. Twenty-four plants were spaced 1 foot apart within a row and rows were 25 inches apart. The clubroot garden, which was in a three-year rotation of grain, hay and swedes, had been inoculated with chopped, clubroot infected roots every third year for many years.

Roots were harvested and classified for clubroot reaction and flesh colour during the second week of September. All data were taken on the basis of individual plants. Each plant in the clubroot garden was removed with a spade and carefully washed so that small infection nodules and lesions would not be lost in pulling or overlooked in classification. Plants were rated for disease reaction on a scale of 1 to 6, 1 being free of clubroot and 6 very severe infection.

Estimates of environmental and genetic variances were obtained in a manner similar to that employed by Powers (3) except that the data, being approximately normal, were not transformed to logarithms.

The methods used in determining the number of major factor pairs which differentiate the parents for resistance and susceptibility to clubroot were similar to those used by Powers, Locke and Garret (4).

The symbols  $P_1$  and  $P_2$  refer to the resistant white fleshed parent and the susceptible yellow fleshed parent, respectively.  $B_1$  refers to a backcross of the  $F_1$  generation to a particular parent, for example  $B_1$  to  $P_1$ .

In the interpretation of data odds as great as, or greater than, 19 to 1 against differences being due to chance were chosen as the level of significance.

## EXPERIMENTAL RESULTS

## 1. The Genic Differentiation of Resistance and Susceptibility to Clubroot

*Homozygosity of Parents*

As is shown in Table 1, the total variance of each of the four  $F_1$  populations studied was significantly different from its estimated environmental variance. This would not be expected if the parents were homozygous for all of the factor pairs which differentiate them in clubroot reaction. For this reason the genetic variances of the populations derived from two parents cannot be accurately determined and a study of genic dominance and genic interactions is prohibited.

TABLE 1.—TOTAL AND ESTIMATED ENVIRONMENTAL VARIANCES OF THE  $F_1$  POPULATIONS STUDIED

| $F_1$ population | Total variance | Environmental variance | F value |
|------------------|----------------|------------------------|---------|
| A $\times$ E     | 0.887607       | 0.700580               | 1.27*   |
| A $\times$ F     | 0.774111       | 0.552451               | 1.40**  |
| C $\times$ E     | 1.436368       | 0.846306               | 1.70**  |
| C $\times$ F     | 0.933483       | 0.628750               | 1.48**  |

\* P. is beyond 0.05.

\*\* P. is beyond 0.01.

Further evidence of the lack of homozygosity of the parents was obtained from a study of the  $F_2$  generation frequency distributions which resulted from two individual  $F_1$  plants of the same cross. As is indicated in Table 2, all comparisons differed significantly with the exception of the comparison between (A  $\times$  F) (b) and A  $\times$  F. The parental progeny frequency distributions are also presented in Table 2. The consistency of reaction of these progenies for resistance or susceptibility, as the case may be, will be noted. In the E(s1), F(s1) and F(s2) distributions a total of fifteen plants were placed in disease classes 1 and 2. These were all found in single plots which had low mean infection values indicating poor soil inoculation with the clubroot organism. A consideration of these facts suggests that the parents were homozygous for major factors differentiating clubroot reaction but that minor factors were segregating.

*A Study of the Factors Differentiating Parents A and E*

The hypothesis which provided the most satisfactory explanation of the number of factor pairs differentiating parents A and E for clubroot reaction was that two major factor pairs were involved, that susceptibility was partially dominant to resistance and that the effects of the genes were additive and had equal effects. The methods of analysis are presented.

If two factor pairs differentiate resistance and susceptibility 6.25 per cent of the  $F_2$  genotypes should theoretically be like that of the susceptible parent. Also, 25.00 per cent of the  $B_1$  to  $P_2$  population should be genotypically similar to the susceptible parent. The actual penetrance of the  $P_2$  genotype into infection class six of the  $F_2$  and  $B_1$  to  $P_2$  populations was 10.3 per cent (0.90/8.70) and 34.3 per cent (2.98/8.70), respectively, as

TABLE 2.—A COMPARISON OF THE FREQUENCY DISTRIBUTIONS OF THE PARENTAL PROGENIES AND OF THE  $F_2$  DISTRIBUTIONS BASED ON THE PROGENIES OF TWO ROOTS FROM INDIVIDUAL  $F_1$  HYBRIDS

| Population | Frequency distribution, infection classes |     |     |     |    |    | Mean infection | Total plants | P. values for chi square |
|------------|-------------------------------------------|-----|-----|-----|----|----|----------------|--------------|--------------------------|
|            | 1                                         | 2   | 3   | 4   | 5  | 6  |                |              |                          |
| A(s1)      | 119                                       | 46  | 2   | —   | —  | —  | 1.31           | 167          | 0.50 - 0.20              |
| A(s2)      | 165                                       | 56  | 6   | —   | —  | —  | 1.30           | 227          |                          |
| A(s1)      | 119                                       | 46  | 2   | —   | —  | —  | 1.31           | 167          | 0.01 - **                |
| C(s1)      | 156                                       | 22  | 2   | —   | —  | —  | 1.15           | 180          |                          |
| F(s1)      | 1                                         | 3   | 13  | 42  | 61 | 15 | 4.58           | 135          | 0.05 - 0.02*             |
| F(s2)      | 3                                         | —   | 5   | 6   | 30 | 11 | 4.40           | 55           |                          |
| F(s1)      | 1                                         | 3   | 13  | 42  | 61 | 15 | 4.58           | 135          | 0.10 - 0.50              |
| E(s1)      | 5                                         | 3   | 26  | 71  | 63 | 16 | 4.29           | 184          |                          |
| (A × E)(b) | 52                                        | 104 | 89  | 64  | 23 | 3  | 2.73           | 335          | 0.01 - **                |
| (A × E)    | 49                                        | 47  | 57  | 52  | 25 | 9  | 3.05           | 239          |                          |
| (A × F)(b) | 41                                        | 98  | 106 | 104 | 67 | 10 | 3.22           | 426          | 0.20 - 0.10              |
| (A × F)    | 32                                        | 103 | 100 | 85  | 38 | 10 | 3.13           | 368          |                          |
| (C × E)(b) | 89                                        | 126 | 106 | 60  | 19 | 2  | 2.55           | 402          | 0.01 - **                |
| (C × E)    | 58                                        | 92  | 117 | 83  | 33 | 7  | 2.94           | 390          |                          |
| (C × F)(b) | 85                                        | 126 | 117 | 83  | 32 | 5  | 2.70           | 448          | 0.01 - **                |
| (C × F)    | 49                                        | 112 | 95  | 85  | 61 | 12 | 3.08           | 418          |                          |

\*\* Highly significant.

\* Significant.

may be seen from an examination of Table 3. These values more nearly approximate those expected on the basis of two factor pair differentiation than if one or three factor pairs are considered.

TABLE 3.—THE FREQUENCY DISTRIBUTIONS, MEANS, AND NUMBER OF INDIVIDUALS STUDIED IN THE POPULATIONS OF THE (A  $\times$  E) GROUP

| Population                | Frequency distribution, infection classes |       |       |       |       |      | Mean infection value | Total population |
|---------------------------|-------------------------------------------|-------|-------|-------|-------|------|----------------------|------------------|
|                           | 1                                         | 2     | 3     | 4     | 5     | 6    |                      |                  |
|                           | %                                         | %     | %     | %     | %     | %    |                      |                  |
| A(s1) = (P <sub>1</sub> ) | 71.26                                     | 27.54 | 1.20  | —     | —     | —    | 1.31                 | 167              |
| B <sub>1</sub> to A(s1)   | 30.53                                     | 38.05 | 20.80 | 6.64  | 3.54  | 0.44 | 2.16                 | 226              |
| F <sub>1</sub>            | 9.13                                      | 19.18 | 23.74 | 35.62 | 12.33 | —    | 3.24                 | 219              |
| F <sub>2</sub>            | 15.52                                     | 31.04 | 26.57 | 19.10 | 6.87  | 0.90 | 2.73                 | 335              |
| B <sub>1</sub> to E(s1)   | 3.57                                      | 14.88 | 19.05 | 36.90 | 22.62 | 2.98 | 3.70                 | 168              |
| E(s1) = (P <sub>2</sub> ) | 2.72                                      | 1.63  | 14.13 | 38.58 | 34.24 | 8.70 | 4.29                 | 184              |

### *Measure of Dominance*

If there is no phenotypic dominance the mean of the F<sub>1</sub> generation should equal the average of the means of the two parents. The difference between these two values was found to be significant as indicated by the "t" test, and, therefore phenotypic dominance probably does exist. The difference between means P<sub>1</sub> and P<sub>2</sub> is 2.98, between F<sub>1</sub> and P<sub>1</sub> is 1.93 and between P<sub>2</sub> and F<sub>1</sub> is 1.05. Therefore the genes carried by the susceptible parent (P<sub>2</sub>) are partially dominant to the resistant parent (P<sub>1</sub>) and the genotypes AAbb and aabb may be assigned to the respective parents. The heterozygous condition of A and B accounts for 64.8 per cent (or 1.93/2.98) of the total difference in infection between AAbb and aabb and 35.2 per cent (or 1.05/2.98) of the difference is due to the added effect of the factors A and B in the homozygous condition.

### *Determination of the Aabb and aaBb and AABb and AaBB Frequency Distributions*

In order to substantiate the hypotheses which were advanced concerning the gene action of the two parents, it first becomes necessary to determine the theoretical frequency distributions of the various genotypes which compose the F<sub>2</sub> population.

The theoretical penetrance of the F<sub>1</sub> and P<sub>1</sub> genotypes into each disease class of the B<sub>1</sub> to P<sub>1</sub> population may be obtained by multiplying the corresponding obtained percentages of rows 6 and 7 of Table 4 by the theoretical per cent of the genotype expected in the B<sub>1</sub> to P<sub>1</sub> population, that is, by 25 per cent in both cases. Row 2 of Table 4 is obtained by summing these values. The theoretical frequency distribution of the remaining genotypes, Aabb and aaBb, as determined from the B<sub>1</sub> to P<sub>1</sub> population, is given in row 4 and is expressed on the basis of 100 per cent in row 5.

In a similar manner, as is shown in rows 8 to 13 of Table 4, the theoretical frequency distributions of AABb and AaBB genotypic individuals may be obtained from the B<sub>1</sub> to P<sub>2</sub> population.

TABLE 4.—PARTITIONING THE FREQUENCY DISTRIBUTIONS OF  $B_1$  TO  $P_1$  AND  $B_1$  TO  $P_2$  POPULATIONS OF THE ( $A \times E$ ) GROUP INTO THEIR COMPONENT GENOTYPES

| Population and genotype                                              | Row number in table | Frequency distribution, infection classes |                |               |              |              |           | Theoretical genotypic per cent |
|----------------------------------------------------------------------|---------------------|-------------------------------------------|----------------|---------------|--------------|--------------|-----------|--------------------------------|
|                                                                      |                     | 1                                         | 2              | 3             | 4            | 5            | 6         |                                |
| $B_1$ to $P_1$<br>$F_1 + P_1$ (AaBb aabb)                            | 1                   | %                                         | %              | %             | %            | %            | %         | %                              |
|                                                                      | 2                   | 30.53<br>20.10                            | 38.05<br>11.68 | 20.80<br>6.24 | 6.64<br>8.90 | 3.54<br>3.08 | 0.44<br>— | 100.0<br>50.0                  |
| Row 1—Row 2<br>Row 3 on positive bases<br>Aabb + aaBb (100 per cent) | 3                   | 10.43                                     | 26.37          | 14.56         | —2.26        | 0.46         | 0.44      | 50.0                           |
|                                                                      | 4                   | 10.43                                     | 26.37          | 13.20         | —            | —            | —         | 50.0                           |
| $F_1$ (AaBb)<br>$P_1$ (aabb)                                         | 5                   | 20.86                                     | 52.74          | 26.40         | —            | —            | —         | 100.0                          |
|                                                                      | 6                   | 9.13                                      | 19.18          | 23.74         | 35.62        | 12.33        | —         | 25.0                           |
| $B_1$ to $P_2$<br>$F_1 + P_2$ (AaBb + AABb)                          | 7                   | 71.26                                     | 27.54          | 1.20          | —            | —            | —         | 25.0                           |
|                                                                      | 8                   | 3.57                                      | 14.88          | 19.05         | 36.90        | 22.62        | 2.98      | 100.0                          |
| Row 8—Row 9<br>AABb + AaBB (100 per cent)                            | 9                   | 2.96                                      | 5.20           | 9.47          | 18.55        | 11.64        | 2.18      | 50.0                           |
|                                                                      | 10                  | 0.61                                      | 9.68           | 9.58          | 18.35        | 10.98        | 0.80      | 50.0                           |
| $F_1$ (AaBb)<br>$P_2$ (AABb)                                         | 11                  | 1.22                                      | 19.36          | 19.16         | 36.70        | 21.96        | 1.60      | 100.0                          |
|                                                                      | 12                  | 9.13                                      | 19.18          | 23.74         | 35.62        | 12.33        | —         | 25.0                           |
|                                                                      | 13                  | 2.72                                      | 1.63           | 14.13         | 38.58        | 34.24        | 8.70      | 25.0                           |

*Determination of the AAbb and aaBB Frequency Distributions*

A knowledge of the theoretical means of the various genotypes occurring in the  $F_2$  population is required in order to estimate the frequency distributions of the AAbb and aaBB genotypic individuals which occur in the  $F_2$  population.

Since 3.70 is the mean infection value for the  $B_1$  to  $P_2$  population, the minimum total infection required for the population in order to have each of the four genotypes represented is 4 (3.70) or 14.80. The mean of the AAbb and AaBb genotypic individuals which occur in the population are obtained from the  $P_2$  and  $F_1$  means, respectively. Therefore, the mean of the sum of the AAbb and Aabb genotypic individuals is  $14.80 - (4.29 + 3.24)$  or 7.27. Since this value involves two individuals the mean of the AAbb and AaBB individuals is  $7.27 \div 2$  or 3.635.

In a similar manner the mean infection value of the individuals of the Aabb and aaBb genotypes may be obtained from the  $B_1$  to  $P_1$  population. This mean is 2.045.

Since  $P_1$  is 1.31 and  $\bar{F}_1$  is 3.24, one A or one B gene in the heterozygous condition adds  $(3.24 - 1.31) \frac{1}{2}$  or 0.965 units to susceptibility. The mean of the  $P_2$  population is 4.29 and, therefore, in the homozygous condition one A or one B gene adds  $(4.29 - 3.24) \frac{1}{2}$  or 0.525 units to susceptibility. Since AAbb differs from Aabb by one A gene and aaBB differs from aaBb by one B gene, the mean of the AAbb and aaBB individuals is calculated to be  $2.045 + 0.525$  or 2.570. This mean is closer to the mean of the Aabb and aaBb genotypic individuals than to that of the  $F_1$  population. Also since in the homozygous condition, one A or one B gene contributes less to susceptibility than does one A or one B gene in the heterozygous condition, the frequency distribution of the AAbb and aaBB genotypic individuals resembles the frequency distribution of Aabb and aaBb more closely than that of the AaBb population. Therefore, by weighting the frequency distribution of Aabb and aaBb by the theoretical expression of dominance of 64.8 per cent and the AaBb frequency distribution by 35.2 per cent a rough estimate may be made of the AAbb and aaBB frequency distribution. The theoretical frequency distributions and the theoretical means of the component  $F_2$  genotypes are presented in Table 5.

A theoretical  $F_2$  frequency distribution may be calculated from the theoretical component distributions. This was done by multiplying the theoretical per cent of a particular  $F_2$  genotype by its respective frequency value in a given class and then summing the values obtained for that class. The theoretical  $F_2$  frequency distribution (Table 5) and the obtained  $F_2$  distribution (Table 3) do not differ significantly as chi-square is 6.448 and  $P$  lies between 0.30 and 0.20.

On the basis of the "t" test where "t" is equal to 1.73 and the degrees of freedom are 334 the theoretical  $F_2$  mean of 2.889 (Table 5) does not differ significantly from the obtained mean of 2.73.

From this study it appears that considerable confidence may be placed in the hypothesis that the parents A and E are differentiated by two major factor pairs for clubroot reaction, that these genes are equal and additive in their effects and that susceptibility is partially dominant to resistance.

TABLE 5.—THE  $F_2$  FREQUENCY DISTRIBUTION OF THE ( $A \times E$ ) GROUP PARTITIONED INTO ITS COMPONENT GENOTYPES ON THE BASIS OF TWO FACTOR PAIRS DIFFERENTIATING THE PARENTS WITH SUSCEPTIBILITY PARTIALLY DOMINANT

| Genotype                       | Frequency distribution, infection classes |       |       |       |       |      | Theoretical $F_2$ genotypic per cent | Mean infection value |
|--------------------------------|-------------------------------------------|-------|-------|-------|-------|------|--------------------------------------|----------------------|
|                                | 1                                         | 2     | 3     | 4     | 5     | 6    |                                      |                      |
|                                | %                                         | %     | %     | %     | %     | %    |                                      |                      |
| AABB                           | 2.72                                      | 1.63  | 14.13 | 38.58 | 34.24 | 8.70 | 6.25                                 | 4.290                |
| AABb }<br>AaBB }               | 1.22                                      | 19.36 | 19.16 | 36.70 | 21.96 | 1.60 | 25.00                                | 3.635                |
| AaBb                           | 9.13                                      | 19.18 | 23.74 | 35.62 | 12.33 | —    | 25.00                                | 3.240                |
| AAbb }<br>aaBB }               | 16.73                                     | 40.93 | 25.46 | 12.54 | 4.34  | —    | 12.50                                | 2.570                |
| Aabb }<br>aaBb }               | 20.86                                     | 52.74 | 26.40 | —     | —     | —    | 15.00                                | 2.045                |
| aabb                           | 71.26                                     | 27.54 | 1.20  | —     | —     | —    | 6.25                                 | 1.310                |
| Theoretical $F_2$ distribution | 14.52                                     | 29.76 | 21.47 | 22.06 | 11.25 | 0.94 | 100.00                               | 2.889                |

*A Study of the Factors Differentiating Parents C and E*

The mean disease values, frequency distributions, and number of individuals studied in each population of the ( $C \times E$ ) group are presented in Table 6.

TABLE 6.—THE FREQUENCY DISTRIBUTION, MEANS, AND NUMBER OF INDIVIDUALS STUDIED IN THE ( $C \times E$ ) GROUP

| Population              | Frequency distribution, infection classes |       |       |       |       |      | Mean infection value | Total population |
|-------------------------|-------------------------------------------|-------|-------|-------|-------|------|----------------------|------------------|
|                         | 1                                         | 2     | 3     | 4     | 5     | 6    |                      |                  |
|                         | %                                         | %     | %     | %     | %     | %    |                      |                  |
| C(s1)                   | 86.67                                     | 12.22 | 1.11  | —     | —     | —    | 1.15                 | 180              |
| B <sub>1</sub> to C(s1) | 36.94                                     | 40.54 | 15.32 | 4.95  | 2.25  | —    | 1.93                 | 222              |
| F <sub>1</sub>          | 9.00                                      | 20.85 | 30.81 | 24.17 | 15.17 | —    | 3.18                 | 211              |
| F <sub>2</sub>          | 22.14                                     | 31.34 | 26.37 | 14.93 | 4.73  | 0.49 | 2.55                 | 402              |
| B <sub>1</sub> to E(s1) | 4.45                                      | 10.00 | 32.22 | 32.22 | 17.78 | 3.33 | 3.64                 | 90               |
| E(s1)                   | 2.72                                      | 1.63  | 14.13 | 38.58 | 34.24 | 8.70 | 4.94                 | 184              |

The methods used in studying these data were similar to those employed for the ( $A \times E$ ) group and the conclusions which can be drawn do not differ.

In studying dominance, it is interesting to note that the theoretical expression of dominance of the double heterozygous condition over the homozygous recessive condition is 64.6 per cent and that of the homozygous dominant over the double heterozygous is 35.4 per cent. Comparable values for the ( $A \times E$ ) group are 64.8 per cent and 35.2 per cent, respectively. It may be that the same genes differentiate resistance and susceptibility in the two groups.

Assuming partial dominance of susceptibility and that two major factor pairs differentiate the parents, the theoretical frequency distributions of the component F<sub>2</sub> genotypes were determined. These distributions are presented in Table 7. The chi square test between the obtained and theoretical F<sub>2</sub> frequency distributions, Tables 6 and 7, respectively, gave a P. value between 0.10 and 0.05 indicating a satisfactory fit.

The "t" value of 2.15, with 401 degrees of freedom, calculated in comparing the obtained and theoretical F<sub>2</sub> mean disease values of 2.55 and 2.726 in Tables 6 and 7, respectively, indicates a significant difference.

*A Study of the Factors Differentiating Parents A and F*

Studies of group ( $A \times F$ ) indicate that two major factor pairs differentiate parents A and F. Unlike groups ( $A \times E$ ) and ( $C \times E$ ), resistance of both factors was partially dominant to susceptibility.

From a study of mean infection values presented in Table 8 it will be seen that the double heterozygous condition theoretically accounts for  $(1.87 \div 3.27)$  or 57.2 per cent of the dominance of the genes determining resistance. The remaining 42.8 per cent is accounted for by the difference between the double heterozygous condition and the homozygous dominant condition. A P. value of less than 0.01 was obtained when the "t" test was applied to the average of the means of the two parents and the F<sub>1</sub> generation. Therefore, phenotypic dominance probably does exist.

TABLE 7.—THE  $F_3$  FREQUENCY DISTRIBUTION OF THE  $(C \times E)$  GROUP PARTITIONED INTO ITS COMPONENT GENOTYPES ON THE BASIS OF TWO FACTOR PAIRS DIFFERENTIATING THE PARENTS WITH SUSCEPTIBILITY PARTIALLY DOMINANT

| Genotype                       | Frequency distribution, infection classes |       |       |       |       |      | Theoretical $F_3$ genotypic per cent | Mean infection value |
|--------------------------------|-------------------------------------------|-------|-------|-------|-------|------|--------------------------------------|----------------------|
|                                | 1                                         | 2     | 3     | 4     | 5     | 6    |                                      |                      |
|                                | %                                         | %     | %     | %     | %     | %    |                                      |                      |
| AABB                           | 2.72                                      | 1.63  | 14.13 | 38.58 | 34.24 | 8.70 | 6.25                                 | 4.290                |
| AABb                           |                                           |       |       |       |       |      |                                      |                      |
| AaBB                           | 3.04                                      | 8.76  | 41.96 | 33.06 | 10.86 | 2.32 | 25.00                                | 3.545                |
| AaBb                           | 9.00                                      | 20.85 | 30.81 | 24.17 | 15.17 | —    | 25.00                                | 3.180                |
| AAbb                           |                                           |       |       |       |       |      |                                      |                      |
| aaBB                           | 20.01                                     | 49.07 | 16.99 | 8.56  | 5.37  | —    | 12.50                                | 2.250                |
| Aabb                           |                                           |       |       |       |       |      |                                      |                      |
| aaBb                           | 26.04                                     | 64.54 | 9.42  | —     | —     | —    | 25.00                                | 1.695                |
| aabb                           | 86.67                                     | 12.22 | 1.11  | —     | —     | —    | 6.25                                 | 1.150                |
| Theoretical $F_3$ distribution | 17.61                                     | 30.54 | 23.62 | 17.79 | 9.32  | 1.12 | 100.00                               | 2.726                |

TABLE 8.—THE FREQUENCY DISTRIBUTIONS, MEANS AND NUMBER OF INDIVIDUALS STUDIED IN THE (A × F) GROUP

| Population              | Frequency distribution, infection classes |       |       |       |       |       | Mean infection value | Total population |
|-------------------------|-------------------------------------------|-------|-------|-------|-------|-------|----------------------|------------------|
|                         | 1                                         | 2     | 3     | 4     | 5     | 6     |                      |                  |
|                         | %                                         | %     | %     | %     | %     | %     |                      |                  |
| A(s1)                   | 71.26                                     | 27.54 | 1.20  | —     | —     | —     | 1.31                 | 167              |
| B <sub>1</sub> to A(s1) | 30.32                                     | 38.01 | 18.55 | 9.50  | 3.62  | —     | 2.17                 | 221              |
| F <sub>1</sub>          | 11.58                                     | 36.32 | 30.00 | 16.31 | 5.79  | —     | 2.71                 | 190              |
| F <sub>2</sub>          | 9.62                                      | 23.01 | 24.88 | 24.41 | 15.73 | 2.35  | 3.22                 | 426              |
| B <sub>1</sub> to F(s1) | 6.50                                      | 9.00  | 16.50 | 31.50 | 29.00 | 7.50  | 3.91                 | 200              |
| F(s1)                   | 0.74                                      | 2.22  | 9.63  | 31.11 | 45.19 | 11.11 | 4.58                 | 135              |

In testing the goodness of fit of the theoretical F<sub>2</sub> frequency distribution presented in Table 9 with the obtained F<sub>2</sub> frequency distribution presented in Table 8, a P. value between 0.01 and 0.05 was obtained indicating that the two distributions do not differ significantly. Also, with a "t" value of 1.30 and 425 degrees of freedom, a non-significant P. value indicated that the obtained and calculated F<sub>2</sub> mean disease values did not differ significantly.

These data give good support to the hypothesis that two major factor pairs differentiate parents A and F for clubroot reaction, that the factors of the resistant parent are partially dominant to those of the susceptible parent and that the genes are equal and additive in their effect.

#### *A Study of the Factors Differentiating Parents C and F*

The hypotheses advanced to explain the differentiation between parents C and F were that one, two or three factor pairs were responsible. It was not possible to substantiate any of these hypotheses.

### 2. Flesh Colour Reaction

Data for individual lines grown on disease-free soil and in the clubroot garden were combined to study root flesh colour inheritance since there appeared to be no real difference between the two tests.

The four F<sub>1</sub> lines were white fleshed and seven of the eight F<sub>2</sub> lines segregated to approximately 15 white: 1 yellow. The chi square values obtained for these seven lines indicated a satisfactory fit to this ratio. From Table 10 it will be noted that, in the case of line (A × E) (b), only that data from the clubroot garden gave a good fit to this ratio. The only explanation which can be given for the deviation of this line from the 15 : 1 ratio is that of chance deviation or some error in classification. The chi square tests for heterogeneity among the seven lines which gave a fit to the 15 : 1 ratio for the flesh colour segregation and line (A × E) (b) of the clubroot garden provided a heterogeneity  $\chi^2$  of 9.82 and a P. value of approximately 0.20 indicating that the lines were from a single population.

It may be seen from Table 10 that backcrosses of the four F<sub>1</sub> lines to the yellow fleshed parent gave chi square values which indicate a good fit to a 3 white: 1 yellow ratio. This ratio is in agreement with the expected on the basis of 15 : 1 segregation in the F<sub>2</sub> populations.

TABLE 9.—THE  $F_2$  FREQUENCY DISTRIBUTION OF THE  $(A \times F)$  GROUP PARTITIONED INTO ITS COMPONENT GENOTYPES ON THE BASIS OF TWO FACTOR PAIRS DIFFERENTIATING THE PARENTS WITH RESISTANCE PARTIALLY DOMINANT

| Genotype                       | Frequency distribution, infection classes |       |       |       |       |       | Theoretical $F_2$ genotypic per cent | Mean infection value |
|--------------------------------|-------------------------------------------|-------|-------|-------|-------|-------|--------------------------------------|----------------------|
|                                | 1                                         | 2     | 3     | 4     | 5     | 6     |                                      |                      |
| AABB                           | %                                         | %     | %     | %     | %     | %     |                                      |                      |
| AABb                           | 71.26                                     | 27.54 | 1.20  | —     | —     | —     | 6.25                                 | 1.310                |
| AaBB                           | 19.22                                     | 44.10 | 21.50 | 10.84 | 4.34  | —     | 25.00                                | 2.330                |
| AaBb                           | 11.58                                     | 36.32 | 30.00 | 16.31 | 5.79  | —     | 25.00                                | 2.710                |
| AAbb                           | 8.62                                      | 15.54 | 19.91 | 29.45 | 21.08 | 5.40  | 12.50                                | 3.475                |
| aaBB                           | 6.40                                      | —     | 12.36 | 39.28 | 32.52 | 9.44  | 25.00                                | 4.175                |
| Aabb                           | 0.74                                      | 2.22  | 9.63  | 31.11 | 45.19 | 11.11 | 6.25                                 | 4.580                |
| aaBb                           | 14.88                                     | 23.91 | 19.13 | 22.23 | 16.12 | 3.73  | 100.00                               | 3.106                |
| aabb                           |                                           |       |       |       |       |       |                                      |                      |
| Theoretical $F_2$ distribution |                                           |       |       |       |       |       |                                      |                      |

TABLE 10.— $F_2$  AND BACKCROSS POPULATIONS STUDIED FOR FLESH COLOUR SEGREGATION AND P. VALUES FOR  $\chi^2$  ON COMBINED DATA OF TWO TESTS

| Population                                                 | Number of plants studied |        | P. values for $\chi$ |
|------------------------------------------------------------|--------------------------|--------|----------------------|
|                                                            | White                    | Yellow |                      |
| <i>F<sub>2</sub> populations</i>                           |                          |        |                      |
| (A × E) (b) (1)                                            | 352                      | 46     | 0.01 - **            |
| (A × E) (b) (2)                                            | 315                      | 20     | 0.95 - 0.50          |
| (A × E) (b) (3)                                            | 667                      | 66     | 0.01 - **            |
| (A × E)                                                    | 632                      | 47     | 0.50 - 0.30          |
| (A × F) (b)                                                | 773                      | 42     | 0.20 - 0.10          |
| (A × F)                                                    | 677                      | 56     | 0.95 - 0.50          |
| (C × E) (b)                                                | 746                      | 61     | 0.20 - 0.10          |
| (C × E)                                                    | 786                      | 41     | 0.20 - 0.10          |
| (C × F) (b)                                                | 832                      | 52     | 0.95 - 0.50          |
| (C × F)                                                    | 807                      | 50     | 0.95 - 0.50          |
| Total $F_2$ population                                     | 5920                     | 415    | 0.50 - 0.20          |
| Total $F_2$ population excluding (1)                       | 5568                     | 369    | 0.95 - 0.50          |
| Heterogeneity test for all $F_2$ populations               | —                        | —      | 0.02 - 0.01*         |
| Heterogeneity test for all $F_2$ populations excluding (1) | —                        | —      | 0.20                 |
| <i>Backcross populations</i>                               |                          |        |                      |
| (A × E) × E(s2)                                            | 207                      | 73     | 0.20 - 0.10          |
| (A × F) × F(s1)                                            | 325                      | 103    | 0.20 - 0.10          |
| (C × E) × E(s1)                                            | 110                      | 38     | 0.95 - 0.50          |
| (C × F) × F(s1)                                            | 341                      | 97     | 0.20 - 0.10          |
| Total backcross population                                 | 983                      | 311    | 0.50 - 0.20          |
| Heterogeneity test for all backcrosses                     | —                        | —      | 0.95 - 0.50          |

\* Significant.

\*\* Highly significant.

(1)  $F_2$  population from disease-free soil.(2)  $F_2$  population from clubroot garden.

(3) Total of (1) and (2).

From these data it may be concluded that the production of yellow flesh colour requires the presence of two gene pairs in the homozygous recessive condition.

### 3. Studies of Independence Between Flesh Colour and Clubroot Reaction

Anderson (1) noted that white fleshed turnips were more subject to clubroot than were yellow fleshed ones. At Nappan, in crosses between white fleshed clubroot resistant swede lines and yellow fleshed susceptible lines, there frequently appeared to be a preponderance of the parental types in the segregating generations. For this reason the eight  $F_2$  populations and four backcross populations to the susceptible yellow fleshed parent, Ditmars Bronze Top, grown in the clubroot garden, were tested for independence of these characters.

Infection classes, 1 and 2, 3 and 4, 5 and 6, were combined and calculations were made according to a  $2 \times N^1$  classification as presented by Fisher (3).

None of the studies of individual populations, nor the totals of the  $F_2$  or backcross populations, indicated association between the characters, that is, non-significant chi square values were obtained. The classification of white and yellow fleshed roots into the three infection classes for the total  $F_2$  and backcross populations are presented in Table 11.

TABLE 11.—THE TOTAL F<sub>2</sub> AND BACKCROSS POPULATIONS CLASSIFIED FOR FLESH COLOUR AND CLUBROOT REACTION IN TESTS OF INDEPENDENCE

| Population                              | Flesh colour | Frequency distribution, infection classes |      |     | P. values for $\chi^2$ |
|-----------------------------------------|--------------|-------------------------------------------|------|-----|------------------------|
|                                         |              | 1-2                                       | 3-4  | 5-6 |                        |
| Total F <sub>2</sub>                    | White        | 1177                                      | 1320 | 338 | 0.99 - 0.95            |
|                                         | Yellow       | 86                                        | 83   | 18  |                        |
| Total backcrosses to yellow susceptible | White        | 82                                        | 266  | 146 | 0.20 - 0.10            |
|                                         | Yellow       | 26                                        | 84   | 65  |                        |

From these data it appears that linkage or other associations do not exist between flesh colour and clubroot reaction in the material studied. If such relationships do exist they are of no apparent significance in breeding programs.

#### DISCUSSION

The analysis of groups (A × E), (A × F) and (C × E) suggests that at least four major factor pairs differentiate resistance and susceptibility to clubroot. The following are possible parental genotypes:

##### *Resistant parents*

A = aabbCCDD

C = aabbCCDD

##### *Susceptible parents*

E = AABBCDD

F = a-b-c-d

The hypotheses which were advanced to explain the various reactions are that, two major factor pairs differentiate parents A and E, and parents C and E, and that susceptibility is partially dominant; two factor pairs differentiate parents A and F and resistance is partially dominant to susceptibility to *Plasmodiophora brassicae*. It was not possible to determine a factorial differentiation between parents C and F. From the results obtained, it seems logical to conclude that, in the cross A × F, the F<sub>1</sub> plant selected for study resulted from a combination of gametes abCD and abcd. Since it was impossible to obtain a satisfactory genetic hypothesis for the factors differentiating clubroot reaction in parents C and F it would appear that parent F was heterozygous for minor factors and possibly for some or all of the major factor pairs involved.

It is of some interest to note that, although parent F appears to have greater susceptibility than parent E, the F<sub>1</sub> generations involving parent F are consistently more resistant than those involving parent E. If the hypotheses which have been advanced and the proposed genotypes are correct, this observation seems logical since, in the F<sub>1</sub> progeny, parent E contributes dominance for susceptibility while parent F contributes some recessiveness for susceptibility. Of interest also is the fact that, in the two groups (A × E) and (C × E), the F<sub>1</sub> hybrids indicated a dominance of susceptibility whereas the F<sub>2</sub> populations more closely resembled the

resistant parent. It may be possible to explain this by the fact that selection of non-infected roots to produce the  $F_2$  population was effective in obtaining minor genes for resistance. However, a similar selection was made in the  $F_1$  population of the ( $A \times F$ ) group and the  $F_2$  population more closely resembled the susceptible parent even though resistance was dominant. This variation in results might possibly be explained on the basis of the genotype of the particular  $F_1$  root selected. Reasons other than those expressed here may, however, be responsible for these results.

It has been shown that duplicate factors are involved in the inheritance of flesh colour and that yellow flesh is obtained when the genes are homozygous for the recessive condition. Also, from tests of independence it was found that little, or no, association exists between clubroot and flesh colour reaction in the material studied.

### SUMMARY

The number of major factor pairs differentiating two  $S_4$  clubroot resistant plants of *Brassica campestris* var. *napobrassica* from two susceptible open-pollinated plants, and the inheritance of flesh colour and its association with reaction to *Plasmodiophora brassicae* were studied. The parental progenies,  $F_1$  and  $F_2$  generations and backcrosses to both parents were used. The conclusions drawn refer, in particular, to the material studied under the conditions existing at the location of the experiment.

Two groups indicated that the parents were differentiated in their reaction to clubroot by two major factor pairs where susceptibility was partially dominant to resistance. One group indicated that two factor pairs differentiated the two parents and that resistance was partially dominant. It was not possible to determine the factorial relationship between the parents in the other group studied. Therefore at least four factor pairs seem to differentiate resistance from susceptibility.

Flesh colour was found to be due to duplicate factors and yellow flesh was obtained when the genes were in the homozygous recessive condition.

From tests of independence it was concluded that any association which may exist between flesh colour and clubroot reaction is of no importance in breeding programs.

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# PRE-HARVEST CHEMICAL TOP-KILLING OF LEGUME SEED CROPS<sup>1</sup>

## I. PRELIMINARY STUDIES WITH PETROLEUM OILS, OIL EMULSIONS AND MIXTURES OF OILS WITH POTASSIUM CYANATE

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### INTRODUCTION

Growers of alfalfa, alsike and red clover seed are naturally concerned about efficient, economical methods of preparing these crops for the threshing operation. In areas with short growing season and variable fall weather, the general practice is to swath the maturing plant material to dry in windrows on the ground prior to use of the combine-harvester with pick-up attachment. An alternative procedure involves leaving the crop stand for direct combining after a killing frost. In either case there may be appreciable losses. Unfavourable weather and waste due to the double handling can reduce returns where preliminary swathing is employed. On the other hand there may be major seed losses owing to pod-shattering during the period before natural freeze-drying permits direct combining of a standing crop. Consequently, methods of obviating the disadvantages of these techniques deserve attention. The use of chemicals that might rapidly kill the top growth of the crop and any intermingled weeds without injuring legume seeds or roots suggests itself as a means of avoiding losses and of facilitating direct combining.

Application of top-killers and defoliant to such crops as tomatoes, potatoes and cotton is established in certain parts of the continent where these crops are grown on a large scale. There is very little literature referring to the pre-harvest top-killing of grain and legume seed crops. Available reports (1) from southwestern United States, however, indicate that dinitro-compounds with diesel oil have been applied from the air with considerable success on flax and alfalfa during the past three years.

At the University of Alberta the use of cyanamid products in dusts and in aqueous sprays on legumes has been under test for a similar length of time, and will be the subject of a later paper. The present report is concerned with results from applications of aromatic weed oil alone, or in aqueous emulsion, or in emulsion with potassium cyanate in small quantity as a fortifying agent.

### MATERIALS AND METHODS

Experiments were conducted in the Edmonton area on duplicated, randomized square-rod plots treated using a knapsack sprayer during sunny weather in the latter part of August when first and second "sets" of seed were nearly ready for harvest but while foliage was still green. The stand of alsike clover was relatively light and short (less than ten inches), while the alfalfa (Grimm) was intermediate, and the red clover (Altaswede) plots were quite dense and tall (about twenty inches).

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Preliminary tests with three experimental oils ranging from 14 per cent to 76 per cent aromatic content showed most rapid and efficient killing from the product with highest aromatic content designated as "Imperial Oil QXS 16C". Accordingly this oil was chosen for subsequent experiments.

Where emulsions of oil and water, or oil and potassium cyanate were used, the mixture included 1 per cent wetting agent "Dreft". Various rates used are noted in the tabular summaries of results.

Samples of seed were taken for germination tests but circumstances prevented the making of suitable yield comparisons.

## RESULTS

### *Alsike Clover*

Table 1 gives the main results obtained with alsike clover two years old.

TABLE 1.—ALSIKE CLOVER

| Chemical     | Rate<br>gal./acre | Mean per cent of top-killing |       |                            |       | *Crown-<br>killing | **Per<br>cent of<br>seed<br>germina-<br>tion |
|--------------|-------------------|------------------------------|-------|----------------------------|-------|--------------------|----------------------------------------------|
|              |                   | 5 days<br>after treatment    |       | 12 days<br>after treatment |       |                    |                                              |
|              |                   | Leaves                       | Stems | Leaves                     | Stems |                    |                                              |
| Aromatic oil | 40                | 98                           | 78    | 100                        | 90    | 0                  | 89                                           |
|              | 60                | 100                          | 95    | 100                        | 95    | 0                  | —                                            |
|              | 80                | 100                          | 98    | 100                        | 98    | 0                  | 86                                           |
| Check        | —                 | —                            | —     | —                          | —     | —                  | 91                                           |

\* As judged by inspection of underground parts and observation of regrowth.

\*\* Courtesy Dominion Plant Products Division analysts, Calgary, Alberta. All samples not submitted.

Of the three rates of oil application noted in the table, the lowest was apparently the most satisfactory. Excessive brittleness caused by the thoroughness of top-killing associated with the use of the larger quantities of oil may be a hazard in high winds or cause losses during the harvesting operation. It appears to be an advantage therefore to have a limited amount of green material remaining near the base of the stem.

### *Alfalfa*

Table 2 summarizes the treatments and results of an experiment with second-year alfalfa which had three aims:

1. To test the feasibility of using a smaller "low" rate of oil application than that used with alsike.
2. To determine the effect of providing greater volume of spray material by adding water with oil in emulsion.
3. To assess the value of incorporation of small amounts of the contact herbicide, potassium cyanate (No. X-1, 91 per cent) with oil emulsion.

Obviously the 20-gallon-rate of oil alone was inadequate, and its extension with 80 gallons water produced no improvement, but instead poorer results. Likewise 60 gallons oil in emulsion was no better than 60

TABLE 2.—ALFALFA

| Chemical                                                           | Rate per acre | Mean per cent of top-killing |       |        |       | Crown-killing | Seed germination |
|--------------------------------------------------------------------|---------------|------------------------------|-------|--------|-------|---------------|------------------|
|                                                                    |               | 3 days                       |       | 7 days |       |               |                  |
|                                                                    |               | after treatment              |       |        |       |               |                  |
|                                                                    |               | Leaves                       | Stems | Leaves | Stems |               |                  |
|                                                                    |               |                              |       |        |       | %             |                  |
| Aromatic oil alone                                                 | 20 gal.       | 80                           | 30    | 85     | 50    | 0             | —                |
|                                                                    | 60 gal.       | 100                          | 75    | 100    | 85    | 0             | —                |
|                                                                    | 100 gal.      | 100                          | 75    | 100    | 100   | 2             | 89               |
| Oil in emulsion (10, 20, 60 gal. to 100 with H <sub>2</sub> O = °) | 10°           | 60                           | 10    | 60     | 10    | 0             | —                |
|                                                                    | 20°           | 90                           | 10    | 90     | 15    | 0             | —                |
|                                                                    | 60°           | 100                          | 75    | 100    | 80    | 0             | 87               |
| K cyanate with 2 gal. oil to 100 with H <sub>2</sub> O = cy +      | 1½ lb. cy +   | 0                            | 0     | 10     | 0     | 0             | —                |
|                                                                    | 3 lb. cy +    | 20                           | 0     | 25     | 5     | 0             | —                |
|                                                                    | 6 lb. cy +    | 25                           | 10    | 35     | 15    | 0             | 91               |
| K cyanate with 5 gal. oil to 100 with H <sub>2</sub> O = cy ++     | 1½ lb. cy ++  | 5                            | 0     | 15     | 5     | 0             | —                |
|                                                                    | 3 lb. cy ++   | 15                           | 10    | 25     | 15    | 0             | —                |
|                                                                    | 6 lb. cy ++   | 80                           | 25    | 80     | 25    | 0             | 90               |
| K cyanate alone in water                                           | 1½ lb.        | 2                            | 0     | 5      | 0     | 0             | —                |
|                                                                    | 3 lb.         | 10                           | 0     | 15     | 3     | 0             | 91               |
|                                                                    | 6 lb.         | 20                           | 5     | 30     | 10    | 0             | 89               |
| Check                                                              | —             | —                            | —     | —      | —     | —             | 91               |

gallons oil alone. The latter treatment yielded satisfactory results. Potassium cyanate at the low rates used by itself or with low gallonages of oil was unsatisfactory. It should be pointed out, however, that good results can be achieved with somewhat higher rates of potassium cyanate in water than those tested here as fortifiers.

### Red Clover

Treatments involved in this experiment with second-year red clover were comparable with those outlined above for alfalfa. Table 3 contains the results. Oil alone at 60 and 100 gallons per acre produced effects considered to be satisfactory in preparation for direct combining. The rather poor results from the 20 gallon-rate of oil were attributable in part to unsatisfactory coverage of the dense vegetation in this stand of red clover. Supplementary water to increase coverage with comparable rates of oil in emulsion, however, did little to improve the situation. Again, as was the case with alfalfa, potassium cyanate as a fortifier was relatively ineffective at the low rates used.

### DISCUSSION AND CONCLUSIONS

In considering the results obtained with the three species of legumes certain major differences and similarities appear. The differences in results apply to plant susceptibility to oil injury. Field observations showed clearly that red clover was most easily damaged, while alsike was somewhat less susceptible and alfalfa was distinctly the most resistant.

TABLE 3.—RED CLOVER

| Chemical                                                              | Rate<br>per acre | Mean per cent of<br>top-killing |       | Crown-<br>killing | Seed<br>germina-<br>tion |
|-----------------------------------------------------------------------|------------------|---------------------------------|-------|-------------------|--------------------------|
|                                                                       |                  | 7 days after treatment          |       |                   |                          |
|                                                                       |                  | Leaves                          | Stems |                   |                          |
|                                                                       |                  |                                 |       | %                 |                          |
| Aromatic oil alone                                                    | 20 gal.          | 48                              | 18    | 0                 | —                        |
|                                                                       | 60 gal.          | 97                              | 88    | 0                 | 89                       |
|                                                                       | 100 gal.         | 100                             | 93    | 2                 | 90                       |
| Oil in emulsion (10, 20, 60 gal.<br>to 100 with H <sub>2</sub> O = °) | 10°              | 13                              | 5     | 0                 | —                        |
|                                                                       | 20°              | 48                              | 33    | 0                 | —                        |
|                                                                       | 60°              | 93                              | 60    | 0                 | 92                       |
| K cyanate with 2 gal. oil to<br>100 with H <sub>2</sub> O = cy +      | 2 lb. cy +       | 10                              | 5     | 0                 | —                        |
|                                                                       | 4 lb. cy +       | 15                              | 10    | 0                 | —                        |
|                                                                       | 6 lb. cy +       | 50                              | 15    | 0                 | 94                       |
| K cyanate with 5 gal. oil to 100<br>with H <sub>2</sub> O = cy ++     | 2 lb. cy ++      | 11                              | 5     | 0                 | —                        |
|                                                                       | 4 lb. cy ++      | 20                              | 10    | 0                 | —                        |
|                                                                       | 6 lb. cy ++      | 50                              | 10    | 0                 | 94                       |
| K cyanate alone in water                                              | 2 lb.            | 5                               | 0     | 0                 | —                        |
|                                                                       | 4 lb.            | 10                              | 0     | 0                 | —                        |
|                                                                       | 6 lb.            | 25                              | 5     | 0                 | 93                       |
| Check                                                                 | —                | —                               | —     | —                 | 94                       |

Using the 60 gallon-rate of oil application as basis for interspecific comparisons in Tables 1, 2 and 3 the results for percentage top-killing might suggest that alsike clover was the most susceptible species. This apparent discrepancy from the field observations noted above finds explanation in the fact that the stand of alsike clover was shorter and much less dense than the red clover which was the heaviest of the three crops. Spray coverage was therefore most intensive on the alsike plants.

As regards similarities in results it was quite obvious that:

1. Regardless of species, the minimum amount of oil required to perform satisfactory top-killing was more than 20 gallons per acre.
2. Where emulsions were used, the added water produced no apparent benefits as compared with similar rates of oil applied alone.
3. The results from applications of two to five gallons oil fortified with up to six pounds potassium cyanate per acre were inferior to results obtained with larger quantities of oil alone.
4. Percentages of germination and of plant crown injury indicate that in these respects none of the treatments caused significant damage. Moreover, studies with cyanate products, to be reported later, suggest that under some circumstances in comparisons between swathed and chemically top-killed material the latter practice may increase fall season root reserves.

It appears, therefore, that sufficient oil can be used safely to perform adequate top-killing of alfalfa, alsike and red clover. The chief question, then, seems to be one of cost of spray material. Since the use of water as

an economy measure to extend the coverage of oil in emulsion is of questionable value, further investigation should be directed to the possibilities of reducing costs per acre by means of ground equipment capable of providing thorough coverage with smaller volumes of oils alone or with various adjuvants. In these regards the United States results from aircraft application noted earlier (1) provide encouragement for the hope that similar economy can be achieved under more northerly environmental conditions.

#### ACKNOWLEDGMENTS

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## BOOK REVIEWS

LEAF ANALYSIS. An authorized translation by R. L. Mitchell of "Die Blattanalyse" by H. Lundegardh. Published by Hilger and Watts Ltd., Hilger Division, London, England. viii + 176 pages. 1951. 22s. 6d. net (postage 6d. extra).

The first of the three chapters of this Book is devoted to "The Scientific Principles of Leaf Analysis" and makes up something over one-half of the monograph. In it are covered such phases of the subject as the mechanism of ion uptake by roots, ion antagonism, and "nutrient index" in the leaves. The second section, "The Practical Basis of Leaf Analysis", gives methods of sampling and preparation, touches on chemical methods of analysis, and presents for the first time details of automatic spectrographic equipment. The single "index" values for potassium, phosphorus, and nitrogen, and increases in yield following the application of the appropriate single fertilizers are discussed and a mathematical formula suggested for the relationships. The third section deals with "Application of Leaf Analysis to the Determination of the Manurial Requirement of the Soil".

The factors influencing the mineral composition of leaves (mainly in the oat plant), the variability during the season and from year to year, water relationships, etc., have all been thoroughly examined. The relationship of diagnoses made from leaf analysis to those made from soil analysis is critically evaluated. The result of much fundamental research is the setting up of a few large, clearly separated levels as a basis for diagnosis. Lundegardh's success is illustrated by the replies of farmers in one test—467 diagnoses were designated as good, 68 as moderate, and 1 as bad. Dr. Mitchell is to be thanked for making more readily available an inspiring monograph.

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MAIZE IN THE GREAT HERBALS, by John Finan. The Chronica Botanica Co., Waltham, Mass. Toronto: Wm. Dawson Subscription Service Ltd. 1951. \$3.00.

This attractive book is an historical account of the introduction of maize (corn) into the continent of Europe, as gathered from the herbals published in many European countries during the renaissance period. Maize was reported growing in Europe very soon after the discovery of America; but while the reports of Columbus and other early explorers made frequent mention of maize, they contained nothing definite concerning the exact time and nature of introduction.

It remained for the European herbalists of the renaissance, who had almost 2,000 years of medical tradition behind them, to definitely record the presence of maize in Europe and to illustrate the plant by means of

wood-cuts, in the great herbals of that time. Beginning in the 1530's the herbalists discussed and described European types of maize plants, but it was not until 1570 that an American origin was suggested. Two distinct types of maize were discussed in the herbals, one a flint which was typical of the flints of Eastern North America, and believed by the herbalists to have originated in Turkey (Asia); and the other, a different type, similar to the present-day maize of the Caribbean area, and which they reported to have been introduced from America. The two types were differentiated in the wood-cuts largely on the basis of ear characteristics and the presence or absence of prop roots.

"Maize in the Great Herbals" contains the only modern account of the early history of maize in western Europe and is issued in co-operation with the Missouri Botanical Garden. It represents a fine effort on the part of the author, who was faced with the problem of translating from the several languages in which the original herbals were published. It is of particular interest to botanists as well as all those who are connected in any way with the maize crop, particularly its origin and history. One observation of considerable interest to students of maize in Canada is that which concerns the conspicuous development of prop roots when tropical varieties are moved farther north. This has been noted in Canada. The characteristic is clearly illustrated in the wood-cut taken from the herbal of l'Obel.

It is of interest to note that the research work, of which this book is the result, was sponsored entirely by the Pioneer Hi-Bred Corn Company, Des Moines, Iowa.

—FREDERICK DIMMOCK,

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 Current science. Bangalore, Current science association.  
 The Times review of the British Colonies. London, Times pub. co. Ltd.  
 Acta vitaminologica. Milan, Italy.  
 Direction of international trade. New York, Statistical office of the United nations.  
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 Annals of eugenics.  
 Schweizerische zeitschrift für tuberkulose. Basel, Switzerland.

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